ARCHIVES OF OPHTHALMOLOGY

VOLUME 19 (old series Volume 76) MARCH 1938 NUMBER 3

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LOCAL BLOODLETTING IN OPHTHALMIC PRACTICE

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Local bloodletting is a time-honored remedy which has fallen into disuse in recent years, being replaced by other methods, more in keeping with newer concepts. However, it is a potent remedy, effecting instantaneous relief from pain and influencing the course of the inflammation. It was practiced by the application of leeches, which mode was abandoned as not compatible with the ideas of antisepsis (although I have never heard of or seen any ill effects from the method), so that nowadays one cannot find leeches in a modern drugstore. This led to the development of the "artificial leech," a trephine-like knife which, quickly rotating cuts through the cutis and produces one or several sharp-edged, circular, profusely bleeding wounds. The place for the application of leeches and of the artificial leech is usually the temple; natural leeches were sometimes applied under the eye.

That local bloodletting is no longer in the armamentarium of the modern ophthalmologist was apparent to me a few years ago, when I heard a paper read in which mention was made of the beneficial effects of leeches used under the eye in cases of scleritis or episcleritis. At that time I mentioned that I often used local bloodletting at the site of the condition.

Bloodletting is beneficial for a number of ocular conditions in which congestion is a prominent symptom and for some in which it is not. It is particularly efficient in relieving the following conditions, and I shall describe the mode of my procedure. While I am not the only one practicing bloodletting at the site of involvement, I do not think that it is in general use or that many of my colleagues are cognizant of the method.

INDICATIONS FOR BLOODLETTING

Persistent Episcleritis.—This condition consists of a torpid localized congestion and swelling of the episclera and sclera, lasting from six weeks to several months. It is benign and sometimes painful and has been variously ascribed to rheumatism, gout and tuberculosis. I advise letting it develop fully, and I especially advise that bloodletting be not

mentioned to the patient during his first visit. After the patient realizes the chronic nature of his condition, he will be more willing to submit to the procedure.

Sharp-pointed iris scissors are used. The patient is directed to turn his eye in if the episcleritis is temporal and out if it is nasal. The eyeball may be held with bare index finger and thumb between the separated lids. Cocaine should not be used on account of its vasoconstricting effect, which would prevent profuse bleeding and therefore defeat the object of the operation. The desirability of profuse bleeding is also the reason why the scissors should be very sharp. The scissors should be opened from 6 to 7 mm. wide and directed at a tangent toward the sclera, and with a quick cut a V-shaped wound is produced through the conjunctiva and episclera. No bandage is necessary. This will dispose of episcleritis in most cases. Rarely I find it necessary to repeat the performance after an interval of a week. There is no possibility of cutting through the sclera. Any one who prefers a knife may take a razor-sharp scalpel and with quick cuts (holding the knife lightly between the fingers) make a cross cut through the nodule. I do not believe that there is a possibility of cutting into or through the sclera.

Iritis and Iridocyclitis.—These conditions, no matter what the cause, especially when dark congestion is present, will be favorably influenced by local bloodletting. With the sharp iris scissors, serveral cuts are made on the lower half of the eyeball from 7 to 8 mm. from the limbus. Also, the effect of atropine on the pupil is greatly enhanced by bloodletting.

Chronic Trachoma with Exacerbations.—For the treatment of this condition the upper lid is everted and a very sharp scalpel is lightly drawn over the conjunctiva and tarsus; two or three cuts are made straight across, especially in the more vascular furrow 2 mm. from the edge of the lid. The patient is directed to look down, and the lower lid is slipped under the upper to protect the eyeball. The knife is turned with the edge upward, and several cuts are made blindly as near as possible to the fornix and farther down. The knife must be lightly held and be very sharp. If it were not extremely sharp, it would defeat the object of the procedure (which is profuse bleeding), and scar formation would result. If the operation is correctly performed, no scars result. The lower lid is treated the same way.

Subacute Conjunctivitis.—The same procedure is used for this condition that is used in cases of trachoma. Patients are frequently greatly relieved and demand performance of the scarification. It has always been a mystery to me that a congested conjunctiva not only becomes

pale immediately after a little bloodletting but may remain so for at least several hours. The good effect can be noticed even after two days.

Relapsing Abrasion of the Cornea.—This troublesome condition of the cornea occurs especially when a cinder has been removed by scraping but also after other types of trauma. Relapsing abrasion is known to every practicing ophthalmologist. The underlying pathologic process is obscure, but it seems related to herpes. How obscure it is, is evidenced by the fact that in "Kurzes Handbuch der Ophthalmologie" [1] several pages are devoted to it, after the perusal of which the reader is left with the distinct conviction that one does not know anything certain about it. My teacher Arlt, whose therapy after sixty years is still unexcelled in most instances, recommended that the eye be bandaged for about five days, after which the abrasion usually remains healed until the next recurrence. By some obscure reasoning, considering that the seat of the trouble is the conjunctival layer of the cornea, I tried scarification of the conjunctiva, with brilliant results in most cases. After scarification, the lids are slightly brushed with a 0.25 per cent silver nitrate solution, and the eye is left unbandaged. The graver forms of this condition, associated with blister formation, loosening of the epithelium of the whole cornea and hypotony, do not yield so strikingly to this treatment but are favorably influenced by it.

1. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 4, pp. 389-391.

CLINICAL ANGIOSCOTOMETRY

A NEW METHOD, WITH THE USE OF DIFFERENT CONTRAST TEST OBJECTS

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Foerster and Aubert were the first (1857) to suggest the use of perimetry for clinical and experimental purposes. This method has given many valuable contributions to clinical and experimental ophthalmology, but after a lapse of thirty years it was found insufficient for examination of the defects of the central visual field. Bjerrum and his followers, particularly Rönne, elaborated in detail the methods of campimetric examination of the visual field.

Under the influence of the Bjerrum school, ophthalmologists became accustomed to regard various defects of the visual field detected by perimetric and campimetric examinations as defects of nerve fiber bundles.

It is interesting that first Hess and then Igersheimer forewarned against the use of minute or dot objects for campimetric investigations.

In his special article on dot perimetry Hess discussed the question of the theory of using small test objects. By means of calculations he showed that images of small objects on the retina may be lesser than, or of the same size as, the retinal vessels. He came to the conclusion that with an entirely normal visual field and no defects provoked therein by the lesions of the optic nerve fibers, the irregularities may be of an occasional character. These defects disseminated in the visual field are due to the presence of rather thick arteries and veins in the inner retinal layers, which hinder the striking of light on the layer of rods and cones immediately underlying these vessels.

The most recent American and English literature on campimetric examination contains special warnings on this subject. The authors use 1 mm. test objects at a distance of 2 meters (Evans, Traquair), i. e., such as those against which Hess and Igersheimer warned.

Studying the blindspot, many investigators, beginning with Helmholtz, recorded that this has not only an oval form but also some dented

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From the Hirshman Memorial Central Ophthalmic Institute (Director, E. B. Rabkin).

shoots which immediately disappear. Helmholtz regarded these shoots as physiologic defects of the visual field and negative images of the retinal vessels from the optic nerve. It was only in 1926 that Evans, using on the stereocampimetric minute, almost dotlike objects, obtained a complete negative image of the retinal vascular system. These ribbon-shaped defects of the visual field, or angioscotomas, said Evans, are the result of the light rays not reaching the optic receptors, on account of the presence in the internal retinal layers not only of large vessels but also of perivascular, lymphatic spaces.

Evans (1926) and Magitot (1935) found that angioscotomas seen on the stereocampimeter are larger than the corresponding vessels. Both authors attribute that to the perivascular lymphatic spaces present. Evans and other authors described a series of angioscotometric investigations on normal eyes and on many eyes with pathologic changes. It appears that the eye with pathologic changes gives an extraordinarily variegated picture of angioscotometric changes of the visual field. Although they had noted and described many series of such changes in glaucoma, periphlebitis and other diseases, Evans and Magitot could not find changes which would be characteristic for singular ocular diseases.

The usual campimetric method of examination, recommended by the Bjerrum school, proved useless for examination in cases of defects of nerve fiber bundles in the visual field with narrow angioscotomas, and not sufficiently precise for the defining of the blindspot and its size. When I made examinations of the blindspot and checked results on the campimeter, I observed that the patient generally shows slowness in his answers in cases of disappearance of the test object and also in cases of its reappearance. Hence I came to the conclusion that the size of the blindspot may vary, depending on whether the test object is seen moving centrifugally or centripetally as regards the centrum of the blindspot. In each case the limits of the blindspot are incorrect. It is impossible to find out the individual errors for each person examined; hence the definition of the size of the blindspot with Bjerrum's method appears not sufficiently precise. This necessitates the elaboration of a new method which may make the examination more precise and permit one to make quantitative measurements of the blindspot and of the angioscotomas.

My method is very simple. When an angioscotoma corresponding to a large retinal vessel is measured campimetrically, its physiologic diameter is generally found to be from 5 to 10 mm. If one makes the examination of an angioscotoma with a small test object moving perpendicularly to the direction of the angioscotoma, the image of the test

object disappears at a certain point in the region of the defect of the visual field. But the image of a sufficiently large test object, the diameter of which exceeds the size of the angioscotoma, when moved in the same direction will pass above this ribbon-shaped defect of the visual field and will not disappear. Consequently if one begins the examination with a small test object and gradually takes other objects each with a diameter 1 mm. larger than the preceding object, one may find an object the image of which will disappear in the patient's visual field for only an instant—and reappear at once. The image of the next object, i. e., that with a diameter 1 mm. larger, will not disappear totally in the visual field. As it passes through the angioscotoma only a portion of it will disappear; i. e., the test object will appear to the patient of a lesser size, but there will be no moment when the patient may think that it has disappeared even for an instant.

The size of the test object the image of which is the last one to disappear even for only an instant in the measured angioscotoma represents, beyond doubt, the exact diameter of the angioscotoma on the campimeter.

I elaborated a test object of a special type and a new method for examination of the blindspot and angioscotomas, which highly differs from those in use and described in the ophthalmologic literature; it is based on the principle of campimetric examination by aid of contrast test objects.

ESSENTIALS OF MY METHOD

After the outlines of the blindspot have been checked on the campimeter in the usual way by Bjerrum's method, I make the measurement of the blindspot's diameter by aid of a special test object in the form of a long band of paper 5 mm. wide, enclosed in a black, narrow, flat case. The back side of this band is graduated in millimeters. This band slides freely in the case. Its protruding end serves as a test object, and one may vary its size from 0 to 300 mm. The test object is generally moved so that its image passes through the horizontal diameter of the blindspot in the centrifugal or the centripetal direction. For the first movement one takes a test object 85 mm. long (on condition that the patient's eyes are fixed on the center of the campimeter at a distance of 1 meter). When one moves the test object slowly there is a moment when the image of this object enters the region of the blindspot. The length of the test object appears to the patient to become smaller and smaller as the image of the object gradually penetrates inside the scotoma. The black case remains invisible on the background of the campimeter. If the diameter of the blindspot is larger than 85 mm. a moment will come when the image of the whole white test object will

disappear in a scotoma for an instant. The next moment the patient will say that the image of the anterior end of the test object makes its appearance out of the region of the scotoma. Finally, when the diameter of the blindspot is less than 85 mm., the anterior end of the test object, the image of which is emerging out of the region of the blindspot, will appear on the other side of the scotoma, i. e., before the posterior end of the test object, the image of which is entering the scotoma, disappears. The patient will observe now that the test object, which he had seen formerly by the sidelong glance, appears in the form of two white dots or as short bands with a black space between.

In the case in which the image of the test object 85 mm. long disappears totally in the blindspot, it is necessary to enlarge its length gradually and choose a length such that the image of the object will disappear in the scotoma for only an instant and with its smallest enlargement will disappear no more.

Finally, in the case in which the diameter of the blindspot is less than 85 mm., it will be necessary to diminish the size of the object until one defines the size of the diameter of the scotoma in the aforementioned manner.

The measuring of the blindspot's diameter with a gray test object is performed in the same manner.

After defining the outline of the blindspot, one draws the outlines of the angioscotomas. For this purpose a 3 mm. test object is moved at some distance along the outline of the blindspot, and all places are checked where the patient sees the test object disappearing. Then the same test object is moved not far from each of the checked dots.

Thus are defined the quantity and the direction of the angioscotomas emerging out of the blindspot.

To draw in detail an angioscotoma tree by the Bjerrum method is difficult and painstaking; it is much easier to check only the direction of the angioscotomas, without drawing their size on the campimeter (fig. 1). This can be done only by the method described for the measurement of the diameter of scotomas. In fact, if one tries to move through the diameter of some angioscotoma the images of gradually larger and larger test objects, it will appear that to measure the diameter of an angioscotoma is as easy as to measure the diameter of the splindspot.

The results of such investigations are checked in the form of fractions. The numerator shows the diameter of a scotoma measured with a white test object, and the denominator shows the diameter obtained with a gray test object.

In the Hirshman Memorial Central Ophthalmic Institute are measured: the horizontal diameter of a blindspot (*BS*), the diameter of an

angioscotoma corresponding to one of the large vascular trunks not far from the optic disk (*AS*) (this measurement is generally made at 45 degrees downward or upward from the horizontal meridian3, and, finally, the diameter of an angioscotoma corresponding to a branch of a small macular vessel (*ASm*). These measurements are made in a simple manner. To draw the contour of a blindspot it suffices to define its limits in from six to eight points on the campimeter, moving a small (3 mm.) test object in the centripetal direction as regards the center of a scotoma. Then the same test object is moved from the center of the campimeter toward the periphery on a radius located 45 degrees upward or downward from the horizontal line. At a distance of from 15 to 16 degrees from the center, the test object which is moved on the

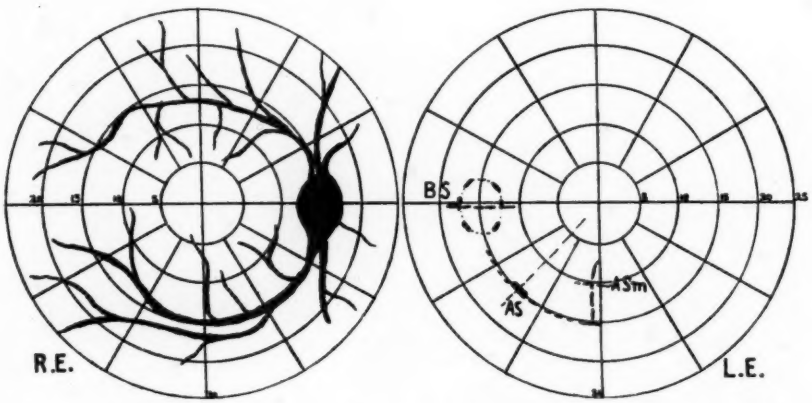

Fig. 1.—The right field shows a normal angioscotometric picture. In the left field the measurement of the blindspot (*BS*) and the angioscotomas (*AS* and *ASm*) by the new method is illustrated.

radius disappears suddenly out of view of the patient. Hence here is located the angioscotoma corresponding to a trunk of a large vessel.

When the test object is moved from the center vertically upward or downward and then at a distance of from 15 to 16 degrees, one may meet the continuation of the angioscotoma which was found formerly.

Now if one moves the small test object from 1 to 2 mm. parallel to the direction of the angioscotoma, it will be possible to find its branches, which run toward the center and correspond to the branches of the macular vessel. The outlines of the macular angioscotomas (*ASm*) are drawn on the campimeter; their measurement is made at a distance of 10 degrees from the center.

The angioscotometric index for the normal eye shows little difference in the values for the numerator and the denominator. This index

for eyes with pathologic changes often shows great variations in the readings for the numerator or the denominator, always toward an increase.

This method would appear not quite complete if it were not supported by proper calculations giving a real picture of the size of the retinal blind strips.

The method of reckoning the diameter of the retinal blind strips according to angioscotograms may be understood from figure 2 (horizontal section of the eye), in which D_1 indicates the fovea centralis; OC_1, radius of 10.2 mm.; E, the nodal point, 6.4 mm. distant from point O (according to Werbitzky's reduced eye); MN, the campimeter; D, the fixation point; ACB, the location of the angioscotoma, showing the size of its diameter, and $A_1B_1C_1$, the location of the retinal blind strip.

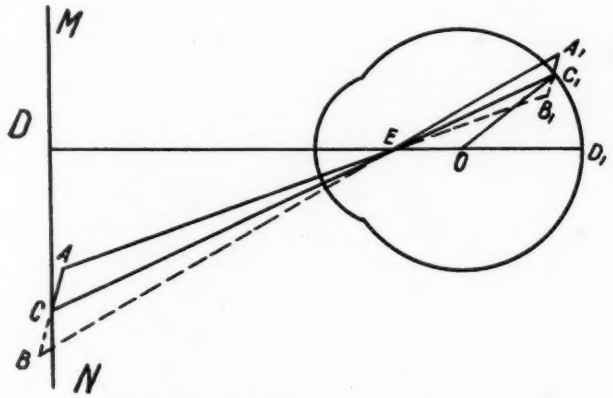

Fig. 2.—Method of reckoning the diameter of the angioscotomas. D_1 indicates the fovea centralis, OC_1, radius of 10.2 mm.; E, the nodal point, 6.4 mm. distant from point O; MN, the campimeter; D, the fixation point; ACB, the location of the angioscotoma, showing the size of its diameter, and $A_1B_1C_1$, the location of the angioscotoma.

The triangles AEB and A_1EB_1 are isosceles triangles with equal angles at the summit E. Thus their bases are strictly proportional to their heights. The base A_1B_1 represents the diameter of the blind strip (to be defined) and equals X; the base of the second triangle, AB, is equal to the size of the smallest white test object (measured in millimeters) which disappears totally for only an instant from the visual field in the corresponding place of the angioscotoma. It remains to define the height of each triangle, CE and EC_1. Figure 2 shows that these values will change in accordance with the angle CED or the distance CD between the scotoma and the center of the campimeter. This is obvious, as it is known that the greater the distance from the center of the campimeter the more the linear size of the degree changes, and that scotomas of practically equal dimensions will appear different,

if one of them is located near the center and the other much farther away. If one recognizes that the distance *ED* from the nodal point *E* of the eye to the center *D* of the campimeter equals 1,000 mm., then

$$EC = \frac{ED}{\cos < CED} = \frac{1{,}000}{\cos < CED} \text{ (formula 1).}$$

As regards *EC*, the reckoning is made according to the formula for the sines of the oblique triangles:

$$\frac{6.4}{10.2} = \frac{\sin < OC_1E}{\sin < OEC_1}; \sin < OC_1E = \frac{6.4 \sin < OEC_1}{10.2};$$
$$< C_1OE = 180° - (< OC_1E + < OEC_1);$$
$$EC_1 = \frac{10.2 \sin < COE}{\sin < OEC_1} \text{ (formula 2).}$$

After *EC* and EC_1 are defined, the reckoning of the width of the angioscotoma in the corresponding place is made according to formula 3:

$$\frac{A_1B_1}{AB} = \frac{EC_1}{EC};$$
$$A_1B_1 = \frac{AB.EC_1}{EC} = AB. \frac{EC_1}{EC}.$$

Assuming that $\frac{EC_1}{EC} = K$, one writes $A_1B_1 = AB.K$ (formula 3),

in which A_1B_1 is the width of the retinal blind strip, *AB* the size of its projection on the campimeter and *K* a coefficient which changes in accordance with the distance of the angle of the corresponding portion of the scotoma from the center of the campimeter.

I reckoned a table of values for *K* and for the size of diameter of the retinal blind strip compared with the size of the test object. The values for the diameters of retinal blind strips are shown in microns. Examination by aid of different contrast test objects will show not only the presence or the absence of the optical function of the retina but also its various stages.

When using a white test object one examines the retina and finds defects of the visual field, after measuring the size of this defect one can say only that the optical function of the retina is absent in the corresponding place.

As regards the condition of the retinal elements localized in the immediate vicinity of the already examined defect of the visual field for white light, such examination gives no results. But when, using my method of examination of an angioscotoma, a blind patch or any other defect of the visual field, one applies not a white but a gray test object, one will naturally obtain greater dimensions of defects. For instance, if when measuring the blindspot one finds that the diameter equals on the campimeter 90 mm. with a white test object and 95 mm. with a gray test object, this will show that the retinal function is lowered at some distance around the optic disk. It is known that in normal eyes a so-called relative blind zone exists around the blindspot; hence it is

obvious that a certain divergence in the results of measuring the blind-spot with white and gray test objects will be a physiologic one. But all readings which surpass the mean values of such divergence will appear to indicate a pathologic condition, and other readings on the campimeter, for instance, 90 and 120 mm., will show the presence of peripapillary retinal inflation.

Likewise, if on examination of an angioscotoma one finds that the size of the white object the image of which disappears in it equals 7 mm. and that of the large object equals from 8 to 9 mm., this small difference will be a physiologic one. But if the corresponding data

TABLE 1.—*Values for Diameter of Retinal Blind Strip Compared with Size of Test Object, Microns*

	Distance from Center, Degrees						
	0	5	10	15	20	25	30
Size of Test Objects, Mm.	Coefficient (K)						
	0.01660	0.01649	0.01619	0.01569	0.01502	0.01417	0.0313
1	16.5	16.5	15.2	15.7	15.0	14.2	13.2
2	33.2	33.0	32.4	31.4	30.0	28.3	26.4
3	49.8	49.5	48.6	47.0	45.0	42.5	39.6
4	66.4	66.0	64.8	62.8	60.1	56.7	52.8
5	83.0	82.5	81.0	78.5	75.1	70.9	66.0
6	100.0	99.0	97.0	94.0	90.0	83.0	79.0
7	116.0	115.0	113.0	110.0	105.0	99.0	92.0
8	133.0	132.0	130.0	126.0	120.0	113.0	106.0
9	149.0	148.0	146.0	141.0	135.0	126.0	119.0
10	166.0	165.0	162.0	157.0	150.0	142.0	132.0
12	199.0	198.0	194.0	188.0	180.0	170.0	158.0
14	232.0	231.0	227.0	220.0	210.0	198.0	185.0
16	266.0	264.0	259.0	251.0	240.0	227.0	211.0
18	299.0	297.0	291.0	282.0	270.0	255.0	237.0
20	332.0	330.0	324.0	314.0	300.0	283.0	264.0
25	415.0	412.0	405.0	392.0	376.0	354.0	325.0
30	498.0	495.0	485.0	470.0	451.0	425.0	396.0
35	581.0	577.0	567.0	549.0	526.0	496.0	462.0
40	664.0	660.0	648.0	628.0	601.0	567.0	528.0
45	747.0	742.0	728.0	705.0	676.0	638.0	594.0
50	830.0	825.0	810.0	785.0	751.0	709.0	660.0

are, for instance, 7 and 17 mm., respectively, this will show that in the course of the retinal vessels there exists a band-shaped defect of the visual field of another kind, namely, that the white test object is differentiated in the denoted retinal zone and the gray test object is not differentiated there on account of the lowered retinal function.

In such cases one can assume the probable presence of perivascular retinal inflation. A much higher dimension of the defects, for instance, 45 mm. with white test objects and 75 mm. with gray test objects, will show that there is retinal inflation along the course of the vessel, linked to the vessel by its origin, and of such a high degree that an absolute band-shaped scotoma is formed. This scotoma is of a considerable width, and at each side there is a relative scotoma, corresponding to the area of lesser intensity of the retinal inflation.

To define the physiologic dimensions of the blindspot Dr. M. L. Kalkutina made, on my request, an angioscotometric examination of one hundred healthy eyes, mostly emmetropic. Only a small number had slight anomalies of refraction. This material was studied by the method of variational statistics. Thus were defined the mean dimensions for the physiologic scotomas of a normal eye. These dimensions were reckoned for three checking points (*BS, AS* and *ASm*) and specially for a white test object. The differences in the results of the angioscotometric examination with gray and with white test objects were reckoned.

The results obtained are shown in tables 2 and 3.

With the use of a table prepared for campimetric examinations at a distance of 1 meter (table 1) it was easy to find out if the obtained mean

TABLE 2.—*Diameter of the Blindspot and of Angioscotomas, Obtained with White Test Objects*

	Mean, Mm.	Variation, ±, Mm.
BS	84.5	6.3
AS	7.1	1.3
ASm	2.3	0.5

TABLE 3.—*Differences in Angioscotometric Readings Obtained by Examination with Gray Test Objects and Those Obtained by Examination with White Test Objects*

	Mean, Mm.	Variation, ±, Mm.
BS	6.6	2.0
AS	2.3	0.5
ASm	1.6	0.3

dimensions of the blindspot and the angioscotomas correspond to the size of the optic disk and of retinal vascular trunks.

With the aid of this table the following results were obtained:

The horizontal diameter of the blindspot equals 85 mm.; this corresponds perfectly with the real dimensions of the optic disk (1.35 mm.). The diameter of an angioscotoma corresponding to one of the large vascular trunks measures approximately 7 mm., and the diameter of this vessel 110 microns. The diameter of the macular angioscotoma measures also approximately from 2 to 3 mm.; hence the caliber of the large macular vessel is from 31 to 45 microns.

According to Hess and other authors, the vascular trunks in the region of the optic disk have variable calibers, but their mean size equals 100 microns.

Thus the mean dimensions obtained for physiologic scotomas perfectly correspond with the quantitative anatomic correlations in the eye. For the blindspot the difference between the limit determined with a

white test object and that determined with a gray test object generally does not surpass from 8 to 10 mm. For the diameter of an angioscotoma and that of the vascular trunk the difference does not surpass from 2 to 3 mm.; for the macular branch this difference generally equals from 1 to 2 mm. Thus, on the basis of the results of the investigations just described, it is possible in the examination of any patient to make, in the first place, deductions concerning the functional conditions of his retina, utilizing for this purpose the diameter of the blindspot and of the angioscotomas, obtained with white and gray test objects.

The investigations were divided into two principal groups. The first group represents experimental investigations for the definition of the cardinal question, namely, the character of the angioscotomas. The second group represents the clinical study of eyes with various pathologic processes and angioscotometric examination of the persons with perfectly normal visual organs but suffering from disturbed circulation of the spinal fluid (high intracranial tension) and circulation of the blood (in stages of perfect compensation and in various degrees of subcompensation or decompensation).

EXPERIMENTAL PART

I selected for my experiments three patients with emmetropic eyes and visual acuity always of 1.

EXPERIMENT 1 (Patient K).—Pressure was exercised on one eye. The diameter of the blindspot of the angioscotomas of the second eye were measured before pressure, during pressure and immedately after pressure until all dimensions of the blindspot and of the angioscotomas returned to their initial extent. When pressure was exercised on one eye, the reactive hypertension in the other eye produced enlargement of the blindspot and of the angioscotomas. Table 4 shows the course of the changes produced by transitory reactive hypertension of the eye.

The blindspot had remained practically unchanged in diameter with the white test object and also with the gray test object. Its enlargement remained extraordinarily slight. The angioscotomas did not undergo considerable changes. The maximal enlargement of the diameters of the scotomas with the white and the gray object appeared seven minutes after the beginning of pressure and remained unchanged for from three and one-half to four minutes until the end of pressure. Three and one-half minutes after the end of pressure the dimensions of the angioscotomas became normal.

EXPERIMENT 2.—To prove the affirmations of various authors as to the presence of an indubitable connection between the lymphatic circulation in the nose and that in the posterior part of the eyeball I repeated the experiment of Evans: the stopping of nasal breathing. The nose of patient K was filled with tampons; she breathed calmly with her mouth open, and I made on the campimeter the measurement of the blindspot and of the angioscotomas. No changes in the diameter of the blindspot occurred (table 5). The angioscotomas showed no enlargement when examined with white test objects and a slight enlargement when examined with gray test objects.

This experiment lasted thirty minutes. Then the nasal breathing was resumed. After seven and one-half minutes the diameter of the angioscotomas showed their initial measurements.

TABLE 4.—*Changes in the Diameter of the Blindspot and of the Angioscotomas in One Eye When Pressure Was Made on the Other (Examination of Right Eye of Patient K)* *

Region Examined	Diameter Before Experiment	Time, Minutes									
		0	2	4	5	7	9	10½	11½	13	14
BS	80/96	Beginning of Pressure on Left Eye	..	..	..	..	85/96	Pressure Discontinued	....	..	80/06
AS	6/12		7/15	..	9/17	9/17	9/17		....	7/15	6/12
ASm	2/6.5		..	4/10	..	6/14	6/14		3/9	..	2/6

* The dimensions of the angioscotomas corresponding to branches of smaller macular vessels (ASm) and those corresponding to larger vascular trunks (As) showed on the day of the determinations (rhinitis subacuta) enlargement for the gray test object. Since the limits for the white test object remained normal, this did not hinder the experiment.

TABLE 5.—*Changes in the Diameter of the Blindspot and of the Angioscotomas with Stopping of Nasal Breathing (Examination of Right Eye of Patient K)*

Region Examined	Diameter Before Experiment	Time, Minutes													
		0	3	5	8	12	15	26	31	33	34	36	38	39½	41
BS	80/96	Beginning of Stopping of Nasal Breathing	....	....	....	...	80/96	....	80/96	Unobstructed Nasal Breathing	....	...	....	....	80/92
AS	6/12		6/13	6/15	6/18	...	.....	6/18	6/18		6/16	...	6/15	6/13	6/12
ASm	2/6.5		....	2/8	....	2/9	.....	2/9	2/9		....	2/8	2/7	2/7	2/6

TABLE 6.—*Changes in the Diameter of the Blindspot and of the Angioscotomas with Total Stopping of Breathing (Examination of Right Eye of Patient K)*

Region Examined	Diameter Before Experiment, Mm.	Breathing Stopped for 30 Sec.	Time After Breathing Was Resumed, Seconds									
			5	15	30	40	50	60	70	80	90	100
BS	4	7	6	..	..	5	..	..	..	..	4	..
AS	2	6	..	5	..	..	4	..	3	..	..	2
ASm	1	4	..	3	3	..	..	2	..	..	1	..

EXPERIMENT 3.—To produce congestion in the eye I totally stopped the patient's breathing for thirty seconds. The measurement of the angioscotomas was made between twenty-five and thirty seconds. On account of lack of time no measurements of the blindspot were made, but only those of the angioscotomas at three points. Their enlargement was marked. Then breathing was resumed, and the moment was defined when the diameter of the angioscotomas became normal at all the points of observation. As it was not possible to make the investigation with more than one test object, I used only the white test object.

In all three points the dimensions of the angioscotomas became normal after from ninety to one hundred seconds. Then I investigated only the blindspot also for thirty seconds, during the stopping of breathing, and defined the moment when its dimensions became normal. It was measured with white and gray test objects (table 7).

EXPERIMENT 4.—I made one more experiment by interrupting the return of the venous blood from the head. I put the cuff of a Riva-Rocci apparatus on the neck of the patient and pumped air. This pressure (from 70 to 75 mm. of mercury) produced considerable reddening of the face.

The retention of the venous blood occurred at the cost of the compression of the skin and jugular veins. Subjective changes occurred: difficulty in breathing, palpitation of the heart, the sensation of a rush of blood to the head (a feeling as if the head was bursting) and temporal pulsation. No subjective visual changes

TABLE 7.—*Changes in the Diameter of the Blindspot with Total Stopping of Breathing*

Test Object	Time Before Experiment	Breathing Stopped for 30 Sec.	Time After 55 Sec.	Time After 65 Sec.
White	90	96	90	90
Gray	98	105	..	98

TABLE 8.—*Changes in the Diameter of the Blindspot and of the Angioscotomas in Cases of Hampered Reflex of Blood from the Head*

Region Examined	Diameter Before Experiment	Time, Minutes											
		0	2½	6	7½	9	12	13	13½	14½	15	16	18	20
BS	80/96	Beginning of Pressure on Veins of Neck	....	....	....	....	80/96	....	Pressure Discontinued	....	...	....	....	80/96
AS	6/12		6/15	6/16	6/20	....	6/20	....		6/18	...	6/16	6/13	6/12
ASm	8/6.5		3/7	....	....	3/10	3/10	3/11		3/10	2/8	2/7	2/6.5	2/6.5

were noted. This experiment showed that the size of angioscotomas became changed also, but not to such a degree as in the preceding experiment. Their diameters remained unaltered when examined with white test objects; changes were observed by examination with gray test objects.

The size of the blindspot remained unchanged. This demonstrates the fact that total interruption of breathing causes much greater congestion than interruption of the return of the venous blood from the head by means of compression of the superficial veins.

Further control experiments, made by special methods, gave very interesting results.

It is known that when one sits in a sulfur bath (H_2S) important changes occur in the system of cardiac vessels. In consequence of expansion of the superficial vessels intense reddening of the skin immediately appears. Its line of limitation is generally located between the parts of the body immersed in water and the outer parts. When the

subject comes out of the bath one often sees on the buttocks, on the parts of contact when sitting, two white patches on a red background. These are the parts of the body which did not come in contact with sulfurated hydrogen (H_2S). During such a bath there occur an afflux of blood into the peripheral sections of the blood vessels and a reflux from the interior organs and the brain.

Three and one-half minutes afterward the skin generally turns paler. The peripheral vessels commence to contract, and the inner organs receive the blood which during bathing flowed out into the peripheral vessels.

EXPERIMENT 5.—My aim was to trace the changes in the retinal vessels under the influence of a sulfur bath (H_2S). For this purpose I used the Gullstrand ophthalmoscope with a modification, namely, placing in the ocular tube of a special prism with 3 mm. openings in its center. This permitted me to make a fairly precise measurement of the caliber of the vessels and to express the data in microns.

When I examined the eyeground I saw two images, one through the opening in the prism and the other produced by the light rays which passed through the prism and were refracted at an angle corresponding to the strength of the prism.

As I already knew the refraction power of the prism and the magnifying power of the apparatus, I was able to measure the caliber of the vessels with sufficient precision.

I fixed an arrow on the ocular part of the apparatus and a stationary graduated disk in front of it. Knowing for how many degrees I turned the ocular tube each time, I could reckon the refraction power of the prism. When the vessel being examined appeared as a double one, I turned the prism till the borders of the two images of the same vessel came in contact. At that time the red strip in the ocular field corresponding to the vessel being examined appeared twice as wide as a single vessel. The location of the arrow was marked in degrees on the disk. Then one image of the vessel was transferred to the other side of the principal image and once more drawn together with the latter till their borders again came into contact, but on the other side. Then the location of the arrow was again marked. The angle of refraction divided into halves gives the corresponding value of the angle the sine of which equals the caliber of the vessel (fig. 3). Then I made the necessary reckonings in accordance with the refraction power of the prism and the magnifying power of the apparatus. One may not make such reckonings for the comparative values; the caliber of the vessel may be shown in degrees of the angle of refraction of the prism.

The experimental patients, B. and C., were placed in a sulfur bath (H_2S), and the measurement of the vessels was made before entering the bath and after coming out of the bath. Table 9 shows objectively in degrees of the angle of refraction of the prism considerable dilatation of the retinal veins and arteries under the influence of the sulfur bath (H_2S).

I also made angioscotometric examinations of these patients. A small campimeter was fixed on the bath; a board with a special rest for the chin of the experimental patient was put across the rims of the bath; the patient was placed in the empty bath, and an accurate angioscotometric measurement was made. Preliminary examination was made under the usual laboratory conditions. The results obtained were analogous.

Thereupon the bath was half filled with water and a sulfurated hydrogen compound put into it. Angioscotometric examinations were made every minute. The results are shown in table 10.

Two minutes after the patients entered the bath the angioscotomas of each were considerably contracted; this was attributed to the diminishing of the caliber of the retinal vessels. This contraction lasted only six minutes. Four and one-half minutes later the retinal vessels of the first patient, and, after almost the same period, those of the second, showed the initial dimensions of their calibers. The dilatation of the vessels appeared rather soon and was of a high degree.

Ten minutes after taking the bath each patient was examined under laboratory conditions with the usual campimeter and the white and gray test objects.

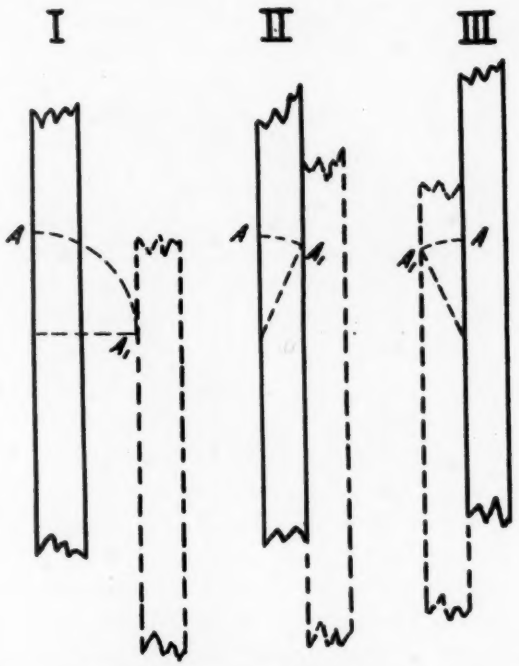

Fig. 3.—Method of measuring the caliber of retinal vessels. *I* shows two images obtained with a prism. *II* shows the borders of the images brought into contact. *III* shows the images brought together on the opposite side. The angle of refraction divided into halves gives the value of the angle the sine of which equals the caliber of the vessel.

After the bath the examination of the first patient was continued during seven, and that of the second patient during five, hours. The blindspot and the angioscotomas showed a tendency to diminish in dimension only after an interval of five hours.

It is interesting that during approximately two hours the difference in the results of the examination with white and gray objects was 0 or slightly higher; afterward this difference became greater and lasted until the end of the examination.

An explanation of this fact may be that on account of protracted dilatation of the vessels and their greater porosity the permeability of the walls becomes greater, there is greater interchange between the vessels and the adjacent tissue, and the transudation is prevalent. The presence of fluid behind the walls of the vessels in the perivascular

TABLE 9.—*Changes in the Diameter of the Caliber of the Retinal Veins and Arteries Produced Under the Influence of a Sulfur (H_2S) Bath, Degrees*

	Patient B.					Patient C.				
	Degrees Before Entering Bath	Time After Coming Out of Bath				Degrees Before Entering Bath	Time After Coming Out of Bath			
Vessel		21 Min.	3 Hr., 15 Min.	4 Hr., 15 Min.	5 Hr., 15 Min.		1 Hr., 30 Min.	3 Hr.	5 Hr.	6 Hr.
Vein.....	15	21	20	21	15	25	30	26.5	27	26
Artery.....	10	15	15	14	11	12.5	13.5	17.5	16.5	15.5

TABLE 10.—*Changes in the Diameter of the Blindspot and of the Angioscotomas Produced Under the Influence of a Sulfur (H_2S) Bath*

Patient B

Region Examined	Before Experiment	Time When Patient Was Sitting in Bath				Time After Coming Out of Bath								
		2 Minutes	3 Minutes	4 Minutes	6 Minutes	3 Minutes	4 Minutes	5 Minutes	17 Minutes	30 Minutes	1 Hr., 10 Min.	2 Hours	3 Hours	4 Hours	5 Hours
BS	88/98	..	..	..	..	..	..	..	90/100	90/100	92/105	100/115	97/110	100/115	95/110
AS(1)†	15*	..	..	..	..	..	..	..	.....	22	30	40/42	40/50	40/50	35/45
AS(2)†	7*	4	4	4	4	4	5	6	8	10	12	23	20/25	18/22	17/20

Patient C

Region Examined	Before Experiment	Time When Patient Was Sitting in Bath				Time After Coming Out of Bath				
		2 Min.	3 Min.	4 Min.	6 Min.	25 Min.	1 Hr., 20 Min.	2 Hr., 40 Min.	4 Hr., 40 Min.	6 Hr., 50 Min.
AS (1)	6*	6	6	5	4	12	15	20/25	30/37	28/35
AS (2)	5*	4	4	4	3	8	8	8/11	9/13	9/13
ASm	3*	3	2	1	1	5	5	5	5	4

* White test object only.
† AS(1) is AS determined on the radius of the campimeter located 45 degrees upward, and AS(2) is AS determined downward from the horizontal line.

space accounts for the appearance of the wide scotomas for gray test objects.

Comment.—The results of the angioscotometric examination and of the measurement of the retinal vessels with the Gullstrand ophthalmoscope and my device were identical. These experimental data corroborate the statements of Evans, Samoylov and Magitot that the angioscotomas are the shadows of the retinal vessels and the lymphatic perivascular spaces.

CLINICAL PART

In accordance with Evans, Dimshitz and Magitot, I observed even in my first investigations that in certain diseases the changes of the blindspot and the angioscotomas are expressed in an extraordinarily high degree. I examined during 1935-1936 over six hundred patients with ocular diseases of various etiology. I made these investigations chiefly in cases of glaucoma, retrobulbar neuritis, neuroretinitis, choked disks, increased intracranial tension with suspected tumor of the brain and diseases of the meningeal membranes. In all these cases the blindspot and the angioscotomas showed great changes. In patients with glaucoma I always found an enlarged blindspot (Bjerrum's scotoma) and enlarged angioscotomas. To ascertain if glaucoma really appears as a factor producing enlargement of the blindspot and of the angioscotomas I applied the pilocarpine test and measured the angioscotomas from every half hour to one every hour. The statement of Samoylov on the specific character of this test for glaucmoa was corroborated. After the instillation of pilocarpine hydrochloride into the glaucomatous eye the dimensions of the blindspot and of the angioscotomas appeared diminished when examination was made with white and gray test objects. In normal eyes and in cases of other diseases the instillation of pilocarpine hydrochloride does not produce such an effect.

The advantage of the pilocarpine test used with my angioscotometric method is that the normal dimensions of the blindspot found with white test objects may be much larger when examined with gray objects. Thus, in cases in which the blindspot appears of normal size as examined with the usual methods, scotometry with the use of different contrast test objects reveals enlargement. In such cases the positive result of the pilocarpine test indicates the presence of glaucoma of the prodromal stage. The parallel examination of the dimensions of the angioscotomas in one or two points completes the examination and helps one to make a diagnosis in dubious cases.

PATIENT R.—During the three months previous to examination the visual field of this patient, aged 56 years, had grown dim. Vision of the right eye was 1, and that of the left eye was 0.1. The visual fields were not contracted. The anterior part and transparent media of each eye were normal. The eyeground of the right eye was normal; that of the left eye showed excavation. There was gray atrophy of the optic disk. The intra-ocular tension of the right eye was 28 mm. of mercury, and that of the left eye was 55 mm. Dark adaptation was lowered in each eye. The results of angioscotometric examination combined with the pilocarpine test are shown in table 11.

The diagnosis was glaucoma of the prodromal stage of the right eye and chronic glaucoma of the left eye.

PATIENT X.—This patient, aged 47 years, had a rather asthenopic condition which might indicate symptoms of glaucoma of the prodromal stage. Vision of each eye was 1. The eyegrounds were unchanged. The visual field showed

great distance between isopters (as determined with 10 and 3 mm. white test objects) in the upper nasal part (in the right eye in a greater degree than in the left eye). In the right eye the blindspot and the angioscotoma were enlarged. Dark adaptation of each eye was normal. The results of the Valsalva test were doubtfully positive for the right eye and negative for the left eye. The results of angioscotometric examination combined with the pilocarpine test were positive for the right eye and negative for the left eye.

In diseases of the optic nerve and on account of the inflammations of the tissue of the nerve trunk, the central retinal vessels are often contracted. First the perivascular lymphatic spaces become contracted;

TABLE 11.—*Results of Angioscotometric Examination Combined with the Pilocarpine Test (Patient R)*

	Before Pilocarpine Test	After Pilocarpine Test					
		½ Hr.	1 Hr.	1½ Hr.	2 Hr.	2½ Hr.	3 Hr.
				Right Eye			
BS	100/120	88/105	78/95	70/88	70/80	74/92	85/105
AS	20/35	15/27	11/24	10/18	12/15	15/22	16/30
ASm	10/18	6/11	4/8	3/6	3/7	6/10	9/16
				Left Eye			
BS	135/175	120/150	110/135	98/20	85/105	88/115	95/135
AS	35/55	25/35	28/30	18/25	13/20	16/25	20/35
ASm	12/25	8/18	6/12	5/10	4/10	5/14	10/20

TABLE 12.—*Results of Angioscotometric Examination Combined with the Pilocarpine Test*

	Before Pilocarpine Test	After Pilocarpine Test			
		1 Hr.	2 Hr.	3 Hr.	3½ Hr.
			Right Eye		
BS	100/125	84/97	74/90	70/80	78/92
AS	30/60	22/46	15/25	10/13	15/25
ASm	7/17	4/2	3/10	4/13	6/14
			Left Eye		
BS	95/100	95/100	95/100	95/100	95/100
AS	8/10	7/9	7/9	7/9	7/9
ASm	2/4	2/4	1/4	2/4	2/4

this produces lymphatic congestion. Slight congestion in the region of the optic disk caused by external squeezing (pressure) on the nerve or by inflammation of the nerve trunk suffices to disturb the lymphatic circulation in the lymphatic spaces and to produce lymphatic congestion along the course of the retinal vessels. The congestion, i. e., the retinal inflation, is shown at once on the campimeter by enlargement of the diameters of the angioscotomas when measured with gray test objects.

Thus angioscotometry with different contrast test objects permits one to define the earliest stages of a lymphatic disorder in the posterior part of the eyeball. But this examination and the produced enlargement of the blindspot and of the angioscotomas are still insufficient for a diagnosis.

When such examinations are complemented with a study of the visual field (by perimetric, campimetric or stereocampimetric examination) and there are found changes in the form of concentric contraction of the visual field in cases of retrobulbar neuritis or in the form of a central scotoma associated with axial neuritis, one may often with a perfect assurance diagnosticate axial or peripheral retrobulbar neuritis, even if no changes in visual acuity are present.

PATIENT SH.—This patient, a woman aged 31 years, suffered from influenza. Some days later she noted hazy vision in the right eye, and a disagreeable feeling. Acuity of vision of the right eye was 1; the eyeground was normal. Perimetric examination showed concentric contraction of the visual field. Campimetric examination revealed that the blindspot and the angioscotomas were enormously enlarged. The diagnosis was perineuritis retrobulbaris postgripposa. Further observation corroborated this. After two and one-half months the symptoms ceased and the patient was discharged.

PATIENT O.—This patient, a woman aged 26 years, showed no changes in the left eyeground, but visual acuity of the left eye was reduced to 1/10. Visual acuity of the right eye was 1. The limits of the right visual field, as determined with 10 mm. and 3 mm. test objects, were normal. There was slight divergence of the isopters. The visual color field was normal. The blindspot and angioscotomas were not enlarged.

Clinical examination of the left eye showed only reduced visual acuity—1/10. Functional examination revealed that the left visual field was concentrically contracted, with considerable divergence of the isopters. In correspondence with the contraction of the visual field, the field for color was also contracted, but only slightly. The blindspot was enlarged, its dimensions being from two to three times larger than normally. There were rather large angioscotomas.

The patient returned after some days. Visual acuity of the left eye was 0.7. She said that on the first day of her visit she had a second attack of influenza. Functional examination showed increase of visual acuity of the left eye and a normal visual field. The blindspot was greatly diminished in size. The angioscotomas were considerably contracted, their dimensions being almost normal.

The diagnosis was perineuritis retrobulbaris gripposa.

In patients with papillitis of various etiology, as in cases of retrobulbar neuritis, enlargement of the blindspot and of the angioscotomas were found. Such changes were observed also in cases of choked disks. Therefore the angioscotometric method cannot serve as the only means of differential diagnosis between choked disks and inflammation of the optic nerve (papillitis and retrobulbar neuritis).

When patients are directed by neuropathologists for examination of the eyegrounds, angioscotometric examination appears to be a great help in cases of increased intracranial tension.

When in cases of refractory and protracted headaches, with a normal picture of the eyegrounds, visual acuity of 1 and a total absence of changes in the field, one finds enlargement of the blindspot and of the angioscotomas, or even normal dimensions as determined with white test objects, and one finds increased dimensions as determined with gray

test objects, one might, almost with perfect certainty, make the statement that the intracranial tension is increased.

Of course, many other factors which may cause enlargement of the blindspot and of the angioscotomas must be excluded. A large percentage of the conclusions regarding, and the diagnosis of, increased intracranial tension by my associates and me were afterward corroborated by neurologic and roentgenologic examinations. This is illustrated by the following cases:

PATIENT K.—During many months there had been refractory and severe headaches. There was total absence of any decrease of visual acuity or changes in the eyeground and of any convincing data based on the neurologic examination. However, examination showed concentric contraction of the visual fields. The increased intracranial tension was corroborated by neurologic examination some time afterward.

PATIENT M.—This patient, aged 22 years, suffered from influenza and constant headaches. The internal organs were normal, as were the accessory sinuses. Visual acuity was 1. The eyeground of each eye was normal. The visual fields were normal. The blindspots and angioscotomas were enormous.

The diagnosis was increased intracranial tension caused by meningitis serosa postgripposa chronica.

The neuropathologic diagnosis was meningitis serosa chronica. Roentgenologic examination showed also increased intracranial tension. A large number of patients who were directed to the ophthalmic institute from clinics for patients with psychoneurologic and nervous conditions associated with proved tumor of the brain showed without exception enlargement of the blindspot and of the angioscotomas, and a great difference in the dimensions of the blindspot and of the minute macular branches when measured with white and gray test objects.

I examined with Dr. S. S. Scheinkman many rheumatic children with deranged circulation of the blood. Complete clinical examination of these children was made by a pediatrician, a rhinologist, a roentgenologist and an ophthalmologist. All the patients with deficiency of circulation of the blood showed enlargement of the blindspots and of the angioscotomas. The dimensions of these scotomas became smaller after treatment.

In cases of insufficiency of circulation of the blood a rise of the hydrostatic tension in the venous part of the capillaries was present (Strazhesko). The products of water metabolism are not entering or almost not entering into the blood. One may expect diffuse inflation of the retina. But this does not occur in patients with symptoms of decompensation or subcompensation; I always found only enlargement of the blindspot and of the angioscotomas. In his article on the pathogenesis of the inflation Strazhesko said that the lymphatic system generally starts to substitute for the reflux of the vascular system when the function of the latter is deranged. It is thus obvious that in cases of noncompensated derangement of the circulation of the blood perivascular and peripapillar retinal inflation is met with.

In this way angioscotometric examination in many cases shows the changes of the physiologic scotomas. Enlargement of the blindspot and of the angioscotomas may be present in cases of glaucoma, diseases of the optic nerve, increased intracranial tension, derangement of the circulation of the blood and other conditions. The change of the angioscotometric index can be observed even in the initial stages of these diseases.

Hence it is obvious that the method of angioscotometry with different test objects, which I proposed, cannot be used as an independent method for functional diagnosis. It is a valuable auxiliary method and may be very helpful when one is considering all the findings of the clinical examination with the aim of excluding a series of factors causing enlargement of the blindspot and of the angioscotomas.

SUMMARY

The proposed new method for the measurement of the blindspot and of the angioscotomas permits one to make a more precise quantitative judgment of the changes in the scotomas than it was possible to do formerly. Exact measurement of the angioscotomas is made possible for the first time.

When measuring the dimensions of the physiologic scotomas by this new method, it is not necessary to make detailed sketches of their contours; this method permits prompt sketching of the outline of the blindspot and of the angioscotomas.

Angioscotometry with the use of a combination of different contrast test objects transforms the proposed method into a clinical one, which defines the functional condition of the retina in peripapillar and perivascular spaces. It permits one to know at once if there is a normal or a pathologic correlation between the blood and lymphatic systems and the retinal tissue.

It is possible by this new method to find pathologic correlations in cases in which with the usual Bjerrum's campimetry a blindspot of normal dimensions is observed.

Angioscotometric examination combined with the pilocarpine test has great advantages over the usual method; it offers a greater possibility of obtaining positive results in cases of glaucoma of the prodromal stage.

The application of different contrast test objects permits one to make a more detailed study of the functions of the eyes in cases of derangement of the circulation and of the lymph of the blood. This method is of importance not only in ophthalmology but also in general medical practice.

Finally, it permits study of the influence of various pharmacologic remedies on the circulation of the blood and lymph of the eye.

PRECANCEROUS MELANOSIS AND DIFFUSE MALIGNANT MELANOMA OF THE CONJUNCTIVA

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The fact that tissue may undergo certain changes prior to the development of a malignant growth is well known. A precancerous stage of varying duration has been observed in the experimental production of epithelioma in animals. In human beings such instances are afforded by the senile dystrophy of the skin which manifests itself by atrophy, degeneration of elastic tissue, flecks of pigment, telangiectasis and circumscribed keratosis. The same changes are seen in xeroderma pigmentosum. Other conditions in which such changes occur are leukoplakia, extramammary Paget's disease and Bowen's disease. Among these rarer manifestations can be mentioned also melanosis of the skin and of the conjunctiva.

In dermatology this is recognized as a precancerous change, and considerable is known about its clinical and histologic characteristics. It may appear in the skin anywhere over the body but has a predilection for the face. It is a rather diffuse nonelevated pigmentation in an otherwise normal appearing skin. It is of a granular appearance, the granules becoming confluent to form more or less dense sites of pigmentation. The average time between the appearance of this melanosis and the occurrence of malignant changes in it is about ten years. The period may be longer or considerably shorter, so that at times the melanosis becomes malignant almost ab initio. The average age at which it appears is 40 years. Miescher [1] reported that of thirty-five malignant melanomas of the skin, fifteen arose from a nevus, ten from normal skin and ten from precancerous melanosis. A similar precancerous melanosis may occur in the conjunctiva and be the forerunner of a diffuse malignant melanoma. Eight such cases are reported here.

Read in part at the meeting of the American Ophthalmological Society, Hot Springs, Va., June 3, 1937.

From the Institute of Ophthalmology and the Memorial Hospital for the Treatment of Cancer and Allied Diseases.

1. Miescher, G.: Geschwülste der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1933, vol. 12, pt. 3, p. 1048.

REPORT OF CASES

CASE 1.—F. R. L., a man, noticed at the age of 45 some pigment spots in the bulbar conjunctiva of the right eye between 7 and 8 o'clock and only a few millimeters from the limbus. The largest area measured 2.5 mm. in the vertical and 2 mm. in the horizontal diameter. There was no elevation. In addition, there were other diffuse poorly demarcated lightly pigmented granular appearing areas, particularly below between 6 and 7 o'clock. The conjunctival vessels leading to the aforementioned areas were somewhat congested. Three years later the main lesion was excised, as it had become slightly larger. Microscopic examination showed diffuse precancerous changes with questionable signs of an early malignant melanoma. Pigmented lesions continued to manifest themselves in this region (fig. 1 *A*), so that two years later irradiation was given, but to no avail. Another local excision was performed, and microscopic examination of a section showed a diffuse malignant melanoma along with precancerous changes of the contiguous conjunctiva. Recurrences followed (fig. 1 *B*). Exenteration was advised, but the patient would not give his consent. The lesion continued to extend, so that in three more years it had spread diffusely over the entire bulbar conjunctiva and most of the palpebral conjunctiva without producing any appreciable elevation or localized tumor formation (fig. 1 *C*). Exenteration was finally done. In four more years papilledema appeared in the left eye, and a relative scotoma developed over the temporal portion of the visual field. The patient died in several months, thirteen years after the appearance of the conjunctival melanosis and eight years after the onset of the malignant growth.

Summary.—There was a diffuse precancerous melanosis of the bulbar conjunctiva for five years prior to the development of a diffuse, almost flat, malignant melanoma, involving ultimately the entire bulbar and palpebral conjunctivae.

CASE 2.—W. W., a man, noticed at the age of 39 a black spot near the inner corner of the right eye. Ten months later, because this spot had increased in size and occasioned some pain and redness, he consulted Dr. John M. Wheeler. In the bulbar conjunctiva of the right eye, between the caruncle and the limbus, was a slightly elevated brownish black mass. The lesion was mainly in the horizontal meridian but extended for some distance superiorly and inferiorly. Surrounding this darkly pigmented tumor was a nonelevated brownish zone of pigmentation. There were three indefinite brownish areas on the nasal aspect of the superior tarsus. The tumor involving the bulbar conjunctiva was excised, and microscopic examination showed a malignant melanoma with precancerous changes in the adjacent conjunctiva. One month later a preauricular gland was noted. The conjunctival tumor spread rapidly, so that in four more months it involved the bulbar conjunctiva almost around its entire circumference and was beginning to appear on the palpebral conjunctiva. In still another two months the tumor occupied the entire circumference of the eye and had extended down into the corneal epithelium from above. There was some bleeding from the tumor. Exenteration of the orbit was done. In six months there were a local recurrence in the outer wall of the orbit and a bluish nodule in the upper skin flap. In two months cervical nodes were palpable, and the orbital recurrence was increasing in size. In one year there were marked loss of weight and pain over the fourth and fifth lumbar vertebrae, which, together with neurologic signs,

indicated spinal metastasis. The patient died two months later, two years and four months after the onset of the condition.

Summary.—There was precancerous melanosis of the bulbar conjunctiva for ten months prior to the development of a diffuse malignant melanoma. The precancerous changes and the melanoma ultimately involved the entire bulbar and palpebral conjunctivae.

CASE 3.—F. I. McB, a man, at the age of 47 first noted a black spot on the temporal bulbar conjunctiva of the left eye. For ten years this remained unchanged, but during the past year it increased in size, and for this reason the patient consulted Dr. John M. Wheeler. Examination showed a dark pigmented mass on the temporal aspect of the bulbar conjunctiva that was roughly triangular in shape with the base at the limbus from 1 to 5 o'clock and extending for a short distance on to the cornea. The apex pointed temporally and ended 12 mm. from the limbus at 3 o'clock. There was some melanosis of the bulbar conjunctiva adjacent to the tumor. There were dilated tortuous conjunctival blood vessels extending to the tumor. Local excision was done one month before the writing of this report, and microscopic examination showed a diffuse malignant melanoma with diffuse precancerous changes in the surrounding conjunctiva. The patient is well and no preauricular or cervical nodes are palpable.

Summary.—There was precancerous melanosis of the bulbar conjunctiva for ten years prior to the development of a diffuse malignant melanoma, with diffuse precancerous changes in the surrounding conjunctiva.

CASE 4.—J. McG., a man, at the age of 67 first noted a pigmented lesion of the right bulbar conjunctiva nasally. Six weeks later he consulted Dr. Franklin Bracken, who found a lesion measuring 6 mm. vertically and 4 mm. horizontally between 2 and 4 o'clock. It was elevated about 2 mm. There was a nonelevated melanosis of the adjacent conjunctiva extending upward as far as 12 o'clock and downward as far as 6 o'clock. Dr. Bracken did a wide excision of the lesion, including the surrounding melanosis. A section of this specimen was not available for examination. After three years a recurrence appeared at 12 o'clock and at 6 o'clock. Excision was done at these two sites by Dr. Bracken, and microscopic examination showed diffuse precancerous changes with localized areas of malignant growth. Dr. James Ewing concurred in the results of the examination. Because of the patient's age, it was elected to try irradiation. The patient was referred to Dr. James Duffy, of the Memorial Hospital, where irradiation with radon in a glass bulb was given to the right eye in the following doses (millicurie minutes with no filter in contact with the lesion):

Date	Mc. Min.
5/24/35	100
7/ 7/35	300
11/20/35	250

The treatments resulted in some regression of the lesions as evidenced by a decrease in the pigment content, but after one year new sites of growth appeared, and during the following year these increased in size and number, so that at the time of the writing of this report there are at the limbus, at 7 and 9 o'clock, flat pigmented areas measuring 2 mm. at the base along the corneal margin and

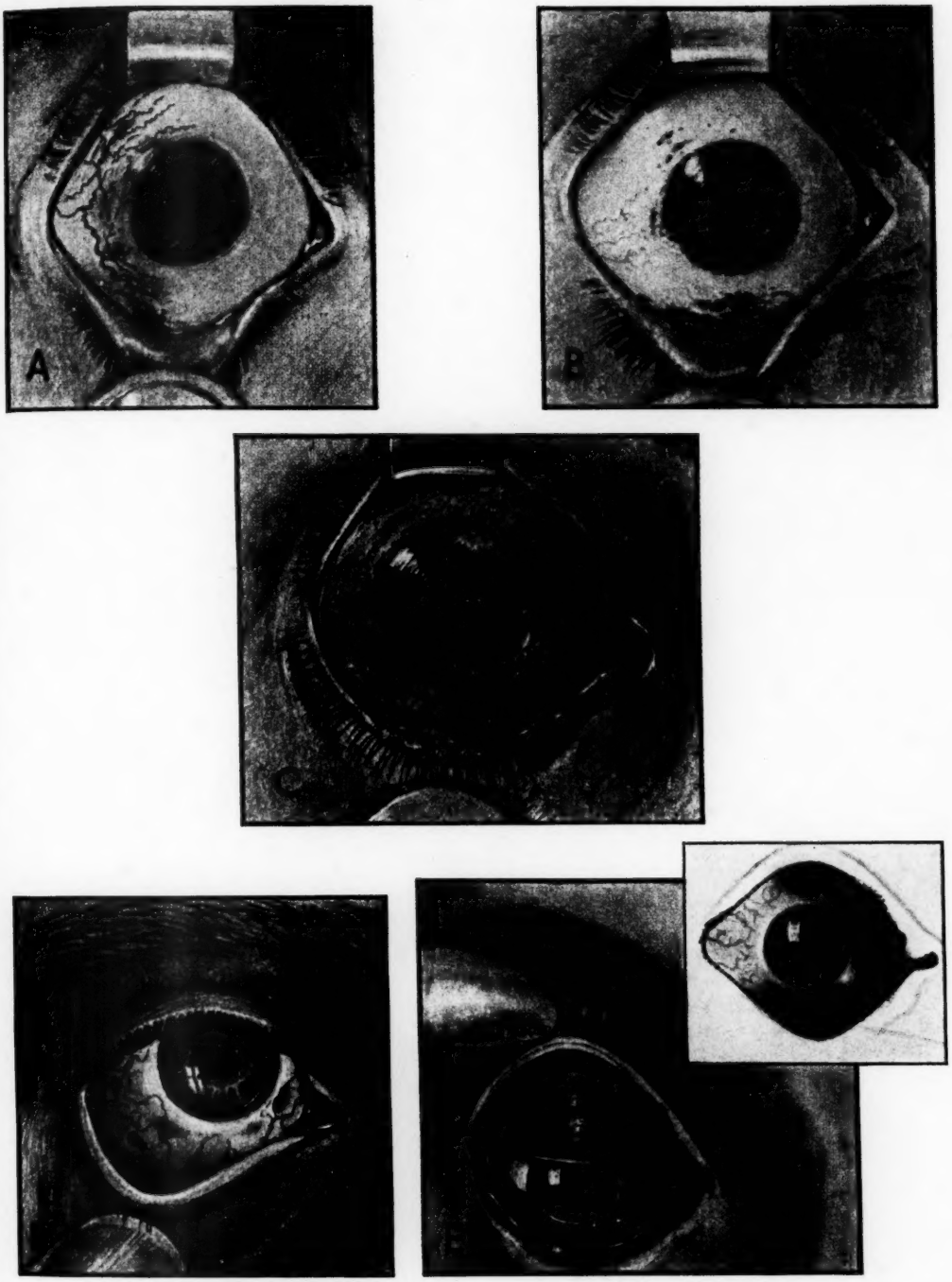

Fig. 1.—*A* (case 1), recurrence after the first excision. *B* (case 1), recurrence after the second excision. *C* (case 1), final extension covering the entire bulbar and palpebral conjunctivae and a portion of the cornea above. The lesion was perfectly flat throughout, with no localized tumor mass at any site. The drawing was made just before exenteration. *D* (case 5), appearance of the lesion just before biopsy and exenteration. *E* (case 8), drawing made with the upper and lower lids everted. The insert shows the extensive involvement of the bulbar conjunctiva. Except for the small localized tumor mass in the lower lid, just nasal to the midline, the lesion showed no elevation.

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extending for 1 mm. out into the surrounding bulbar conjunctiva. Above, in the bulbar conjunctiva, at 11 o'clock and from 8 to 10 mm. from the limbus, are several diffuse flat pigmented areas. The patient is well, and there is no evidence of metastasis.

Summary.—A diffuse malignant melanoma developed apparently spontaneously in the bulbar conjunctiva. There was precancerous melanosis around the original malignant site, and it is possible that the melanosis preceded the malignant changes but was unobserved by the patient.

CASE 5.—K. W., a man, at the age of 54 first noted a nonelevated irregularly shaped pigmented spot on the bulbar conjunctiva of the right eye 8 mm. from the limbus at about 9 o'clock. The lesion enlarged slightly, so that three years later, when it was about 2 mm. in size, it was excised. A section of this specimen was not available for study. The patient's attention was again called to this area three years later by the appearance of two pigmented spots in the region where the former one had been excised. These spots had enlarged slightly during the preceding few months. Examination at the Memorial Hospital showed (fig. 1*D*) two jet black nonraised areas of pigment on the bulbar conjunctiva temporally at between 8 and 10 o'clock and about 3 mm. from the limbus. These measured 2 by 1.5 mm. and 2 by 0.5 mm. There was a smaller lesion above these at about 11 o'clock which measured 2 by 0.5 mm. In the lower portion of the bulbar conjunctiva were several faint light brown diffuse areas of pigmentation. There was no elevation of any of the lesions. The lesions on the bulbar conjunctiva at 8 and 10 o'clock were excised. Microscopic examination of these showed a diffuse precancerous melanosis with signs of an early malignant melanoma in places. The microscopic report was concurred in by Drs. James Ewing, Fred Stewart and Purdy Stout. Exenteration of the orbit was done, and a Thiersch graft was implanted. At the time this report is written, four years after exenteration, the patient is well and there is no evidence of metastasis.

Summary.—There was a precancerous melanosis of the bulbar conjunctiva for six years prior to the beginning of malignant changes. Exenteration was done, and the patient showed no signs of metastasis or local recurrence after four years.

CASE 6.—J. McP., a man, at the age of 55 first noted a pigmentation about the outer aspect of the upper and lower lids of the right eye around the external canthus. This appeared rather suddenly and was not accompanied by symptoms. He thought that it had shown some regression. Four months from the onset he consulted Dr. Hayes E. Martin, who found an irregular nonelevated pigmented lesion of the skin of the eyelids and the adjacent palpebral conjunctiva at the outer canthus of the right eye. The densest pigmented portion was on the palpebral margin and particularly on the conjunctival side of the lower lid, extending from the outer canthus along the lower lid for about one third of its length. A smaller but less extensive lesion was present in the upper lid along the margin of the palpebral conjunctiva, near the external canthus. The pigmentation of the skin extended for 15 mm. temporally from the external canthus and into the upper and lower lid adjacent to it. There was no elevation of any of the lesion. At a consultation with Dr. James Ewing, it was decided that the lesion was precancerous. The patient insisted on trying irradiation, so this was given by Dr.

Martin to the right eye with radon in a glass bulb in the following doses (millicurie minutes with no filter in contact with the lesion):

Date	Mc. Min.
1/27/34	350 to lower lid
3/31/34	250 to outer canthus
6/23/34	250 to margin of lower lid
7/14/34	440 to skin lateral to external canthus
10/ 6/34	350 to skin at external canthus

The lesion regressed rapidly, so that at time this report is written, three years after treatment, no pigmentation is noted either in the skin or in the palpebral conjunctiva. There remains only the sequela that is expected after such irradiation, namely, the absence of cilia along the lower lid for about one third of its distance from the external canthus, and over the same area some atrophy and telangiectasis.

Summary.—There was a precancerous melanosis of the skin of the lids and adjacent palpebral conjunctiva. This was treated with irradiation before malignant signs appeared, and complete clinical regression ensued. There was no reappearance of the lesion after three years. No tissue was obtained for microscopic examination.

CASE 7.—W. E. B., a man, at the age of 43 first noticed a dark pigmentation of the conjunctiva of the left lower lid, mostly in the region of the fornix. He observed no change for five years, when the lesion began to increase in size rather rapidly. He consulted Dr. Grady Clay, of Atlanta, Ga., who found that the lesion involved the conjunctiva of the lower lid from the skin to the fornix and the contiguous bulbar conjunctiva up to the limbus. The inner canthus, including the caruncle and semilunar fold, was also affected. There was no localized tumefaction but merely a diffuse nonelevated pigmentation. No regional lymph nodes were palpable. The patient was sent to Dr. James Ewing and Dr. James Duffy, of New York, for consultation. They advised irradiation with a radon bulb. This was given by Dr. Duffy to the left eye in a glass bulb in the following doses (millicurie minutes with no filter in contact with the lesion):

Date	Mc. Min.
7/10/33	180 to left lower lid
8/14/33	240 to inner canthus
1/ 8/34	120 to upper lid
4/ 2/34	106 to lower lid, external
4/ 2/34	150 to lower lid, sulcus, two areas
4/ 2/34	80 to upper lid, mesially
12/18/34	125 to lower lid, mesially
12/18/34	125 to lower lid, laterally
6/12/35	175 to sclera, outer
6/12/35	312 to lower lid, two areas
6/12/35	312 to end of tarsal cartilage, two areas
12/ 2/36	240 to bulbar conjunctiva
12/ 2/36	180 to upper lid

The first irradiation was given on July 10, 1933. The following months the pigment in the melanotic areas became less, and at sites where formerly this

pigmentation was solid black it became mottled. The extent of the lesion also seemed to lessen. The area around the inner canthus showed no change. Further irradiation was given on August 14. For five months longer it was thought that regression was taking place. Three months later, however, the lesion had spread to the bulbar conjunctiva above and even to the upper palpebral conjunctiva. Further irradiation was given on April 2, 1934, and during the following eleven months regression was noted in some areas and progression in others, so that at the end of the period Dr. Clay reported a definite increase in the size of the lesion of the bulbar conjunctiva, especially at the limbus temporally where there was some invasion of the cornea. Also, he noted a considerable increase along the superior fornix. In three months more, in June 1935, a preauricular node was noted and removed by Dr. Grove, of Atlanta, Ga. A section of this was sent to Dr. Ewing, who reported it to be a malignant melanoma. Nine months later Dr. Clay reported a further progression of the lesion and particularly the presence of a small tumor mass in the fornix below. Nine months later a small mass appeared in the left side of the neck, the liver was palpable and the patient was losing weight. Two months later the patient died. A summary of the autopsy report, furnished by Dr. James J. Clark, of Atlanta, Ga., follows: "The liver weighed 10,500 Gm. and was quite nodular, and many of the nodules had necrosed. Fungating areas of cancer tissue were present outside of the capsule of the liver. The abdomen was distended with about 2,000 cc. of bloody fluid. Several nodules of tumor tissue were present in the mesentery, and each kidney contained tumor tissue which had erupted through the capsule. There were many enlarged mesenteric and retroperitoneal glands. The lungs, mediastinum and glands of the hilus were free from metastasis. Both eyes were obtained for examination and were normal except for the malignant melanoma of the conjunctiva."

Summary.—There was a precancerous melanosis of the palpebral and bulbar conjunctivae for five years prior to the development of diffuse malignant melanoma. Irradiation was used, with some regression, but metastasis and death ensued six years after the onset of malignant changes.

CASE 8.—N. D., a man, at the age of 48 first noticed a dark discoloration along the inner side of the right lower lid and over the white of the eye nasally. This discoloration resembled that caused by the use of mild protein silver, and attention was called to it by several of his friends. He felt that the lesion remained unchanged for the next three years, when it began to increase in size, and a small lump appeared near the edge of the lid. This prompted him to consult his local physician who removed the lump and gave a single application of radium, lasting six hours, the factors of which are unknown. During the next two years the lesion spread, so that when the patient was seen at the Memorial Hospital (fig. 1 *E*) it was represented by a dense, diffuse, mottled, brownish black, nonelevated pigmentation of the entire palpebral conjunctiva of the lower lid, its margin and the contiguous skin for a short distance. Also, the entire bulbar conjunctiva, except temporally and up, was similarly affected. The palpebral conjunctiva of the upper lid was also involved over the nasal and temporal portions. There was a small bluish black nodule in the lower lid which was about the size of the average chalazion. Regional glands were not palpable. There was marked conjunctivitis present, with considerable purulent discharge. On Oct. 10, 1937, the right orbit was exenterated, and a Thiersch graft from the thigh was inserted.

Summary.—There was a precancerous melanosis of the palpebral and bulbar conjunctivae for three years, after which the malignant growth developed, which in two years involved practically the entire palpebral and bulbar conjunctivae with a diffuse malignant melanoma. The lesion was flat, with no elevation except at one site in the lower lid, where a small nodule developed.

COMMENT

From these eight cases it can be said that the average age at which precancerous melanosis of the conjunctiva [2] is recognized is about 48 years, and the average time which elapses before malignant changes ensue is about five years.

DIFFUSENESS

An important characteristic of the type of malignant melanoma discussed in this paper is its diffuseness. The precancerous changes may extend over a wide area, involving sometimes almost all of the conjunctiva. Malignant areas may make their appearance at any site, and usually at many sites, either simultaneously or at varying intervals in the course of the disease. The malignant areas may remain flat, as in the precancerous stage, or they may be somewhat elevated (from 1 to 3 mm.). If the tumor is allowed to grow unheeded, it may ultimately grow into a localized tumor mass. Often, though, it remains flat or slightly elevated and may involve the entire bulbar and palpebral conjunctivae and terminate fatally by metastasis without even forming an elevated mass. Extensive involvement of the conjunctiva results, not necessarily by spreading by direct continuity from one malignant site but rather from multiple scattered foci of origin which arise at different sites in the diffuse precancerous melanosis.

RELATION TO NEVUS

A nevus of the conjunctiva is not an obligatory precancerous lesion, whereas acquired melanosis ultimately becomes malignant. In my experience many more malignant melanomas of the conjunctiva arise from an acquired melanosis or spontaneously than from a nevus. Those that seem to arise spontaneously may be preceded by an unobserved melanosis. In contrast to the diffuse flat character of the lesion described in this paper, the malignant melanoma arising from a nevus grows more as a localized tumor mass. The melanoma that is said to arise from an acquired nevus rather than from the usual congenital nevus seems, at least in some cases, to belong to the group described here, as acquired

2. Congenital melanosis of the conjunctiva, as seen in highly pigmented races and in melanosis oculi, is not to be confused with acquired precancerous melanosis.

melanosis has been incorrectly termed an acquired nevus. Also, many congenital nevi of the conjunctiva have no pigment, either clinically or microscopically, and may be unobserved. If such lesions take on pigmentation later in life, they may become apparent and therefore be considered acquired.

On the basis of the material afforded by these cases, the histologic changes in precancerous melanosis and diffuse malignant melanoma of the conjunctiva are as follows:

The earliest changes in precancerous melanosis appear in the basal layer. These cells increase in number, become pigmented and are separated from one another by clear spaces, and the individual cells swell or become hydropic (fig. 2 *a*). The swelling gives a large protoplasmic element to the cells, which may be perfectly clear or may show an amorphous granular content, which is often interspersed with varying numbers of pigment granules. Some cells show an enormous globular distention associated with degeneration of the nucleus, as evidenced by its poorly staining granular appearance. Several or many adjacent swollen cells may become confluent, forming clear spaces containing the degenerative products of the involved cells. The protoplasm of the less swollen cells may be finely granular or honeycombed and contain what seem to be small droplets, which simulate those seen in embryonic fat cells or foam cells. The nuclei are usually larger than those of the normal basal cell, and the chromatin may be more rarefied, often with a rather large nucleolus.

These altered basal cells proliferate in some places to only two or more cells thick, while in other places they segregate into clumps or nests (segregation), which invade the submucosa (fig. 2 *b*) and may lose their connection with the basal layer (*Abtropfungsprozess*). These nests may also invade the overlying mucosa (intra-epithelial growth), where they stand out in sharp contrast to the normal mucosal cells because of their clear hydropic protoplasm and a clear cleft or space around the nest (fig. 2 *c*). Individual heavily pigmented cells or lightly pigmented cells with clear protoplasm may be seen in the superficial layers of the mucosa and sharply demarcated from their surroundings. At some sites the altered basal cells invade and entirely replace the overlying mucosa. Small epithelial whorls, with slight cyst formation, may be seen occasionally beneath the mucosa. In the submucosa there are varying numbers of chromatophores representing the phagocytosed pigment which has migrated from the basal cells. In places there is sometimes noted a considerable number of chromatophores beneath a relatively normal appearing nonpigmented mucosa or beneath a thin atrophic mucosa or at a site with little or no mucosa on the surface. These areas apparently represent a regression of the lesion, with the resulting dis-

appearance of the mucosa, which in places has not regenerated or has regenerated with normal or atrophic epithelium, leaving only the phagocytosed pigment in the submucosa as remains of the active process. There is frequently a chronic inflammatory reaction of plasma cells and other types of lymphocytes, principally at the limit of the extension into the submucosa (fig. 2 *d*). An important characteristic of the lesion is its diffuseness. The basal layer over wide areas can show the characteristic precancerous changes which here and there become more

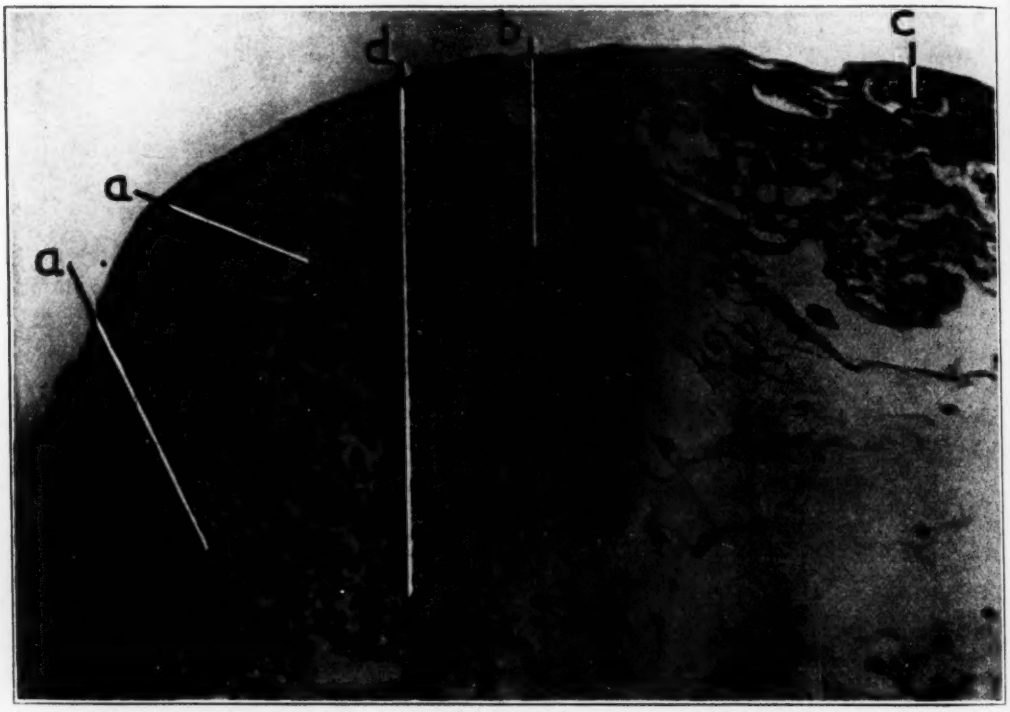

Fig. 2 (case 5).—Photomicrograph of tissue removed for biopsy just prior to exenteration: *a*, the swelling and hydrops of the proliferated basal cells; *b*, the segregation and *Abtropfungsprozess; c*, intra-epithelial growth, and *d*, a mild infiltration of lymphocytes.

advanced and show an increase in the pigmentation, segregation, *Abtropfungsprozess,* intra-epithelial growth, etc. As the lesion becomes malignant, it may still remain diffuse with little localized thickenings, elevation or tumor formation, or there may be tumor formation which is more marked at one or several sites. In a nevus one also sees some of the same characteristics of growth as was noted in these cases, such as segregation, *Abtropfungsprozess* and intra-epithelial extension. There were, however, no neuroid bodies seen or any other evidence to support

a neurogenic origin. I am of the opinion that there is every reason to believe that these tumors arise primarily from the potentially pigment-bearing basal layer of the conjunctiva.

In reviewing the literature on malignant melanoma of the conjunctiva it was often difficult to determine which tumors belonged to the group discussed in this paper, because some of the reports merely described the clinical appearance briefly and frequently gave no data concerning the course, follow-up study or histologic changes. Frequently, it could not be determined whether the lesion had become malignant or, if so, whether it arose originally from a nevus, a precancerous melanosis or spontaneously.

The cases of malignant melanoma of the conjunctiva in which the growth seemed to have developed at the site of an acquired melanosis are those reported by Fromaget,[3] Fano,[4] Dor,[5] Capdeville,[6] Blanch,[7] Roper,[8] Coppes, Capron,[9] Silex [10] and Löhlein.[11] Those in which the growth involved both the skin of the lids and the conjunctiva are reported by Hutchinson [12] and by Lafon and Teulières.[13]

SIGNS OF GROWTH

Besides an increase in size and pigment content, it is stated in the literature that in the cases in which the skin is involved the onset of malignant growth in an area of melanosis is frequently accompanied by signs and symptoms of inflammation. The patient in case 2, reported on here, was prompted to secure medical advice because the lesion had

3. Fromaget: Mélanose conjonctivale et lentigo malin, J. de méd. de Bordeaux **30**:261, 1900.

4. Fano: Mélanose et tumeur mélanique de la conjonctive, Gaz. d. hôp. **45**:651, 1872; Recidive d'une tumeur mélanique de la conjonctive après une première extirpation, ibid. **46**:156, 1873.

5. Dor: Pigmentation mélanique de la conjonctive, Mém. et compt.-rend. Soc. d. sc. méd. de Lyon (1878) **18**:61, 1879; Une malade atteinte de pigmentation mélanique de la conjonctiva et d'une partie de la cornée, Lyon méd. **28**:101, 1878.

6. Capdeville, P. J.: Contribution à l'étude de la mélanose conjonctivale, Thèse de Bordeaux, no. 111, 1911, p. 50.

7. Blanch, A.: Mélano-sarcome de la paupière, Rec. d'opht. **4**:620, 1882.

8. Roper, A. C.: Melanotic Sarcoma of Conjunctiva with Pigmented and Non-Sarcomatous Growths in Skin, Tr. Ophth. Soc. U. Kingdom **32**:117, 1911-1912.

9. Capron, F. P.: Multiple Melanotic Nevi of the Conjunctiva, with Malignant Invasion of the Corneal Limbus, Tr. Am. Ophth. Soc. **14**:322, 1915.

10. Silex, P.: Ueber epibulbäre melanotische Sarcome, Arch. f. Augenh. **20**: 59, 1889.

11. Löhlein, W.: Ueber Melanosis der Bindehaut, Klin. Monatsbl. f. Augenh. **68**:389, 1922.

12. Hutchinson, J.: On Tissue-Dotage, Arch. Surg., London **3**:315, 1891.

13. Lafon, C., and Teulières, T.: Tumeur mélanique primitive de la paupiere, J. de méd. de Bordeaux **38**:536, 1908.

occasioned some pain and was inflamed. In case 8 there was marked inflammation of the conjunctiva with a great deal of mucopurulent discharge. Microscopically, an inflammatory reaction of some degree was noted in most of the cases. Other signs indicating the onset of the malignant growth in cases in which the skin was involved were ulceration and hemorrhage. These occurred in some of the cases in which the conjunctiva was involved, but quite late in the course of the disease.

TRAUMA

In the literature a history of trauma is frequently mentioned, both in the cases in which the skin was involved and in those in which the conjunctiva was involved. The question as to whether the trauma called the patient's attention to a preexisting lesion or whether it was a precipitating factor must be considered. A history of trauma was obtained in only one case reported here. In case 3 the patient said he was hit in the involved eye with a roman candle several years prior to the development of the pigment spot.

SPONTANEOUS REGRESSION

Similar lesions that involve the skin are said sometimes to show areas of regression along with areas of progression. There have occurred authentic cases in which regression has continued to the point of complete disappearance of the lesion. I have never observed this clinically in cases in which the conjunctiva was involved, but I judge, on the basis of the microscopic appearance, that such processes of progression and regression sometimes go hand in hand. Clinically, therefore, this could give a changing picture from time to time. The increase and decrease of pigment in the lesion, as seen clinically, would not necessarily be an accurate criterion. When an area regresses, the conjunctiva disappears; later it may regenerate to an atrophic or relatively normal conjunctiva. The pigment, though, that was produced when the site was active, migrates to the submucosa and remains there in the chromatophores. Therefore, such pigment continues to be seen through the translucent, regenerated and maybe atrophic conjunctiva. Such is probably not the case with the opaque skin.

The patient in case 6 stated that he thought the pigmentation of the skin of the lids had shown some regression prior to treatment. The regression in some of the cases, accredited to irradiation, may have been in part or totally spontaneous.

TREATMENT

Because of the extensive involvement of the conjunctiva, even over areas not appreciable clinically, enucleation is not indicated, as usually

all of the conjunctiva should be removed. Therefore exenteration is necessary, and at the time this is done a Thiersch graft can be placed in the orbit on a gauze tampon. This graft takes well, even on the bare bony surface, and a long course of dressings during the period of granulation is thus avoided. Because of the rather unimportant clinical appearance of the lesion in the early stage, temporizing measures are often employed, and because its diffuseness is not sometimes appreciated, repeated local excisions are done. Much valuable time is thus frequently lost by not doing an early exenteration. Case 5 (fig. 2) was the only one in which exenteration was done early.

In the precancerous stage irradiation may be effective. This is based on the result in one case (case 6) in which the condition seems arrested after three years.

Drs. John M. Wheeler, Hayes E. Martin, James Duffy, Grady Clay and Franklin Bracken furnished me with data relative to patients who were under their care.

The drawings were made by Mr. Gustav Bethke and Miss Emily Freret; the technical work and the preparation of the photomicrograph, by Mr. N. E. Ross and Miss Lily Kneiske.

VITAMINS IN TREATMENT AND PREVENTION OF OCULAR DISEASES

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In this ever changing world the members of the medical profession have found it necessary to treat man's bodily and mental afflictions with newer and more specific therapeutic methods. This evolutionary change has been based on the advancement of science. In the past it was thought that many of the ailments of mankind were produced by changes in the humors of the body. With the advent of newer knowledge of bacteria and their associated toxins, a major portion of the diseases were attributed to bacterial invasion of the body; thus focal infection came into vogue as an underlying cause of many diseases. To this day effort is made to eliminate every possible source of infection in the treatment of inflammations of the various organs, particularly the eye, through eradication of foci of infection, such as the tonsils and teeth, opening of the sinuses and extirpation of many of the other diseased tissues. It has been demonstrated that the local disturbance thus treated is frequently aggravated.

It is obvious, therefore, that supplementary therapeutic measures must be considered in the treatment of some of these refractory diseases. From the experimental work of physiologists and others interested in the subject of nutrition, the members of the medical profession soon learned that the proper selection of food is a most important means of improving and maintaining health. In the light of this knowledge, it seemed advisable to recommend that these stubborn maladies be treated with a well balanced diet supplemented with necessary vitamins.

It has been shown by laboratory research that a specially planned faulty diet may produce definite pathologic changes in the animal body. This type of investigation cannot be duplicated in the clinic for many reasons. Nevertheless, similar dietary deficiencies have been observed among civilians in economic stress, particularly in other countries. It is from their unfortunate experiences and from the few reported cases in this country that it was learned that the incidence of these diseases varies with the climate and the economic status, food supply, age, sex,

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Read at a meeting of the New York Clinical Society of Ophthalmology, Dec. 6, 1937.

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and occupation of the inhabitants. Although typical cases of dietary deficiency are few in the United States, it is now fully appreciated that milder forms of this condition are more prevalent than reported. The incidence of mild avitaminosis as a complication of other disease is estimated as very high. These mild deficiencies may occur not only as independent disturbances but frequently are complications of other diseases. It has been suggested that several mild deficiencies may be present in one person. It is essential to bear in mind that when the intake of food is restricted or when there is an interference with its absorption and utilization a vitamin deficiency may result.

When Osborne and Mendel [1] and McCollum and Davis [2] in 1913 reported ocular inflammations in their respective colonies of animals on a diet free from vitamin A, they opened a new era of nutritional investigation. It remained for McCollum and Simmonds [3] to correlate these observations on rats with those reported by Bloch [4] and Mori [5] in children, and they came to the conclusion that the ocular disease produced experimentally in animals is the analogue of the corneal lesions which these authors reported in man. In view of this finding, they concluded that this type of ocular inflammation is a deficiency disease in the same sense as beriberi and is due specifically to the lack of vitamin A. The work of these pioneers led many investigators, laboratory and clinical, to reinvestigate and to interpret the vast number of previously known ocular disturbances which were reported in the literature. It became apparent that many of these diseases, particularly xerophthalmia and keratomalacia, were in part, at least, produced because of improper food or the lack of a specific vitamin.

It was evident to some observers that xerophthalmia and keratomalacia were late manifestations of vitamin A deficiency, and in 1924 Holm [6] showed that hemeralopia developed early in rats which were kept on a diet free from vitamin A. He also noted that hemeralopia did not result from vitamin A deficiency alone but that it was necessary

1. Osborne, T. B., and Mendel, L. B.: The Influence of Butter Fat on Growth, J. Biol. Chem. **16**:423, 1913.

2. McCollum, E. V., and Davis, M.: The Essential Factors in the Diet During Growth, J. Biol. Chem. **23**:231, 1915.

3. McCollum, E. V., and Simmonds, N.: The Minimum Requirements of the Two Unidentified Dietary Factors for Maintenance as Contrasted with Growth, J. Biol. Chem. **32**:181, 1917.

4. Bloch, G. E.: Eye Diseases and Other Disturbances in Infants from Deficiency in Fat in the Food, Ugesk. f. læger **79**:349, 1917; abstr., J. A. M. A. **68**: 1516 (May 19) 1917.

5. Mori, M.: Ueber der sogenannten Hikan (Xerosis conjunctivae infantum ev. keratomalacie), Jahrb. f. Kinderh. **59**:175, 1904.

6. Holm, E.: Demonstration of Hemeralopia in Rats Nourished on Food Devoid of Fat-Soluble-A Vitamin, Am. J. Physiol. **73**:79, 1925.

to expose the animals to light before it became manifest. The hemeralopia was cured within two or three days by giving the animals an ordinary diet. Sugita [7] likewise demonstrated hemeralopia in rats with vitamin A deficiency. In 1923, however, Fridericia and Holm [8] reported that regeneration of the visual purple after bleaching by light was considerably slower in rats on a diet free from vitamin A than in rats on a normal diet. This observation was also confirmed by Tansley,[9] who followed the course of regeneration of the visual purple in normal rats and in rats deficient in vitamin A. She noted that if the animals were well depleted of vitamin A it took longer for the visual purple to regenerate. In this manner it was shown that the visual purple and hemeralopia were intimately associated in experimental vitamin A deficiency. Later Holm [10] observed a considerable vitamin A content in the retina, and this was confirmed by similar feeding experiments in 1931 by Kriss, Smith and myself.[11] With the aid of the spectroscope, Wald [12] found vitamin A in considerable concentration in solutions of visual purple extracted from the retina and the pigmented choroidal layer of the frog, pig and cattle.

In view of these experiments on animals which showed that hemeralopia was the first demonstrable ocular symptom of vitamin A deficiency, that the content of the visual purple in the retina was disturbed and that considerable vitamin A was present in the normal retina of the experimental animal, the question arose as to whether vitamin A deficiency in man might not also manifest itself, in addition to other symptoms, by varying degrees of hemeralopia, depending on the degree of avitaminosis.

An extensive investigation of hemeralopia was recently made by Frandsen,[13] and the report is very interesting because of her detailed

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7. Sugita, Y.: Experimentelle Untersuchungen ueber die Wirkung der Galle und ihrer Bestandteile auf das Auge, speziell auf den Lichtsinn und den Sehpurpur, nebst Bemerkungen über meine Sehpurpur-Lösungsmethode, Arch. f. Ophth. **116**: 653, 1925.

8. Fridericia, L. S., and Holm, E.: Experimental Contribution to the Study of the Relation Between Night Blindness and Malnutrition: Influence of Deficiency of Fat-Soluble-A Vitamin in the Diet on the Visual Purple in the Eyes of Rats, Am. J. Physiol. **73**:63, 1925.

9. Tansley, K.: Factors Affecting the Development and Regeneration of Visual Purple in the Mammalian Retina, Proc. Roy. Soc., London, s.B **114**:79, 1933.

10. Holm, E.: Demonstration of Vitamin A in Retinal Tissue and a Comparison with Vitamin Content of Brain Tissue, Acta ophth. **7**:146, 1929.

11. Yudkin, A. M.; Kriss, Max, and Smith, A. H.: Vitamin A Potency of Retinal Tissue, Am. J. Physiol. **97**:611, 1931.

12. Wald, G.: Vitamin A in the Retina, Nature, London **132**:316, 1933; Carotenoids and the Vitamin A Cycle in Vision, ibid. **134**:65, 1934.

13. Frandsen, H.: Hemeralopia as an Early Criterion of A-Avitaminosis and Clinical Symptoms and Treatment of This Disease, Acta ophth., supp. 4, 1935, p. 1.

study of the subject. She divided the causes of hemeralopia into two groups, namely, conditions giving rise to changes in the light-refractive apparatus and those giving rise to changes in the light-perceptive apparatus. If the cornea, anterior chamber, lens or vitreous chamber is involved, the light-refractive apparatus is injured; if the retina and choroid are involved, the light-perceptive apparatus is injured. In the latter group Frandsen included a number of different conditions that affect the retina, such as congenital total color blindness and "congenital family hereditary hemeralopia," and malaria, gastro-intestinal disturbances, nephritis, diabetes, anemia, cachexia and hepatic disturbances. Frandsen also pointed out that hemeralopia may be associated with poisoning caused by quinine, carbon disulfide, alcohol, nicotine, epinephrine and various poisonous gases employed in warfare, besides the diseases of the retina and choroid, such as syphilitic choroiditis, disseminated choroiditis, retinitis pigmentosa, detachment of the retina, optic atrophy, optic neuritis, sympathetic ophthalmia, glaucoma and excessive myopia, and Oguchi's disease.

It is not unusual, according to Frandsen, to find a mild degree of hemeralopia during a cursory examination for glasses. The symptoms are so relatively mild that the patient does not seek medical aid. On the other hand, the same symptoms have been severe enough to inconvenience the patient a great deal. Frandsen expressed the belief that there is a considerable difference in the intensity of the symptoms which accompany hemeralopia in men and in women; the symptoms are most pronounced in women at puberty and during the menopause. With a special apparatus she found great individual differences in the hemeralopia of normal school children and of normal adults; also, a number of persons kept on an institutional diet deficient in vitamin A demonstrated hemeralopia of various degree, and patients in private ophthalmologic practice showed hemeralopia much more frequently than other healthy persons.

I am much impressed with Frandsen's views concerning the similarity of the ocular symptoms observed in persons with mild hemeralopia and in those presenting clinical pictures of neurasthenia and hysteria. The symptoms attributed to the latter are asthenopia, poor visual acuity without a definite error of refraction, photophobia, "glare blindness," muscae volitantes, after-images and reduction in the field of vision, which is greater when the patient becomes tired. According to Frandsen, these symptoms may also be present in patients presenting essential hemeralopia.

It may be possible that the same fundamental disturbance of the retina is present in all of these conditions. It is not necessarily a vitamin deficiency. The ocular phenomena in cases of hysteria may be produced

by a disturbance of the perceptive organs of the retina by stimulation with light. It is a known fact that ocular stress in cases of neurasthenia and hysteria is frequently alleviated by the use of plain or tinted lenses and the assurance of relief. I have found the same type of symptoms in patients who cannot be considered as hysteric or neurasthenic or as deficient in the dietary essentials. Most of these persons have blond fundi, and the skin of their body burns when exposed excessively to the sun. Such a person cannot tolerate the rays of the sun or artificial light, has difficulty in driving at night because of the glare of lights but does not experience night blindness. A corrective lens does not always relieve the ocular symptoms. When the patient is given a well balanced diet and rests the eyes when mentally or physically fatigued, the ocular distress disappears and the general health improves.

In view of the similar observations in persons with neurasthenia or hysteria, in those who show definite hemeralopia and in those who have a blonde "make-up," it seems that a method should be found to demonstrate the underlying cause of this ocular disturbance. It is important to decide whether hemeralopia and poor dark adaptation are synonymous, for at present investigators are classifying poor dark adaptation as hemeralopia, and some have gone still further and assumed that poor dark adaptation or hemeralopia is a sign of vitamin A deficiency. I cannot fully subscribe to the latter assumption until further investigation is made. Some of the pharmaceutic brochures are even attributing "glare blindness" to a vitamin A deficiency.

Many types of apparatus and different methods have been used for demonstrating hemeralopia, dark adaptation and "glare blindness;" therefore, it is difficult to compare the various results. At present possibly two instruments may be used in measuring dark adaptation, one devised by Feldman [14] and one devised by Hecht and his associates.[15] Slight degrees of this condition are difficult to diagnose. There are individual variations of dark adaptation, even in healthy persons who have no ocular symptoms. It is apparent that if methods of examination for dark adaptation are to be serviceable in vitamin research they must be easy to manipulate, must not be cumbersome and should be scientific enough for accurate and constant readings. Then clinical observations must be made and norms established. It is important that a decision be made whether poor dark adaptation is the same as hemeralopia and whether the latter is a reliable criterion of vitamin A deficiency. What role do "glare blindness" and retinal fatigue play in this syndrome?

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14. Feldman, J. B.: An Instrument for Qualitative Study of Dark Adaptation, Arch. Ophth. **18**:821 (Nov.) 1937.

15. Hecht, S.; Haig, C., and Wald, G.: The Dark Adaptation of Retinal Fields of Different Size and Location, J. Gen. Physiol. **19**:321, 1935.

RESULTS OF VITAMIN THERAPY

I shall leave this academic discussion and review the results obtained in ocular diseases in which vitamin therapy was employed. At the University Clinic (Yale) many patients with types of ocular disturbances have been treated with vitamins. Many persons with phlyctenular keratoconjunctivitis who were given a well balanced diet, cod liver oil and a standard brand of brewers' yeast became well within one week. These persons did not receive ocular therapy. A number of patients with interstitial keratitis improved rapidly when vitamin therapy was given simultaneously with the local treatment. In one instance the ocular condition improved before the specific therapy was instituted. Frequently there was encountered in children and infants a dryness of the cornea, with similar changes in the epithelium of the conjunctiva, which improved when a diet high in vitamin A was given. Children occasionally appeared in the clinic with a broken-down cornea that was not accompanied by a noticeable ciliary injection. When they were treated systemically (a diet high in vitamin content), the corneal lesion disappeared. Recently my co-workers and I [16] described a similar condition encountered in adults. The patients' ages ranged from 45 to 68 years. A thorough physical examination did not reveal any signs or manifestations of infection; the histories of the patients indicated that the condition was accompanied by loss of appetite, constipation, headache and general malaise. Invariably these patients had lost their teeth early in adult life. The first complaint was that of having something in the eye and photophobia. The cornea appeared normal at first; nevertheless, the eye was extremely painful. The patients noted no visible ocular disturbance. When the eye was examined, there was a breaking down of the periphery of the portion of the cornea which was exposed in the palpebral fissure or in the area covered by the lower lid. There was little if any congestion present. The cornea often stained with fluorescein. The lesion did not seem to improve with local treatment, and within a few days the ulcer appeared to be more extensive. The invaded cornea presented a shallow excavated ulceration, which frequently spread along the margin of the cornea and extended toward the pupillary area. Examination of this area with the slit lamp revealed a swollen, edematous corneal epithelium and a similar involvement of the substantia propria. The surrounding tissue showed considerable vascularity. The corneal nerve fibers extending into the diseased area were prominent.

In this type of corneal lesion, particularly in the early stages, no definite inflammation of the deeper layers of the cornea and uveal tract

16. Yudkin, A. M.; Orten, A. U., and Smith, A. H.: The Production and Cure of Ocular Disturbances in Adult Albino Rats by Adjustment of Vitamin A, Am. J. Ophth. **20**:1115, 1937.

can be detected. Often the lesion is described as a catarrhal ulcer, without any definite inflammation of the conjunctiva. In some instances the ulceration has the appearance of a lesion of the cornea produced by an injury, but no history of injury can be elicited. Frequently, when first seen, the corneal lesion resembles a marginal ulcer, indolent ulcer or rodent ulcer. At this stage the aqueous humor reveals numerous floaters, and Descemet's membrane is sometimes studded with deposits, showing the uvea involved at this stage.

The treatment of this type of corneal lesion has been unsatisfactory. Local medication and heat seem to aggravate and delay the healing process, producing a refractory condition. It is frequently necessary to cauterize the cornea with full strength tincture of iodine or phenol. In this stage the cornea often becomes infected and a hypopyon results, necessitating more drastic treatment.

It was suggested from experience in the laboratory with animals with deficiency diseases that this condition might be a nutritional disturbance; therefore, a number of these patients at the clinic were given cod liver oil in addition to their regular diet. Some showed improvement in a short time, whereas others did not respond. It was evident that some other factor was involved. From the histories of the patients it appeared that the intestinal tract might be at fault. Large quantities of vitamin B complex were given before each meal in addition to the cod liver oil. In the majority of instances the systemic and ocular disturbances disappeared.

Although hemeralopia is the outstanding symptom in retinitis pigmentosa, no definite improvement has been noted when a diet high in vitamin content is employed. I am satisfied that the visual acuity and field of vision in late stages of retinitis pigmentosa are not improved by constant oral administration of potent cod liver oil, its concentrates or the precursor of vitamin A, beta carotene. At the clinic vitamin B complex in large quantities was added to the vitamin A, but the results were the same. The patients were of the opinion that they saw better during the day and found their way about better at night, yet the visual acuity and visual fields, as a rule, showed no improvement on ocular examination. When the neuro-epithelium is destroyed it is difficult to regenerate it. I recommend, however, that this type of therapy be continued, for it is the best there is to offer these unfortunate persons. It is my impression that retinitis pigmentosa if treated early with a diet high in vitamin content may be held in abeyance.

It is remarkable how quickly chorioretinal disturbances alleged to have a focal infection as their underlying cause clear up when the patient is given a well balanced diet enhanced by vitamin A and brewers' yeast powder. It is the policy at the clinic to treat the general condition of the patient simultaneously with the ocular condition. Focal infections

are usually removed, but not until the patient's general health is improved. I have seen too many unfavorable results from disturbing focal infections when the patient's resistance is not too good. Although it has been possible in many instances to cure, check and improve corneal lesions and retinal disturbances by supplementing the diet with vitamin A and B complex, I do not believe that a chorioretinal disturbance should be considered a true vitamin A or B complex deficiency.

VITAMIN B COMPLEX

To one who has been privileged to study many generations of rats in the laboratory and to watch their response to minute changes in the diet, particularly that of the vitamins, there can be little doubt about the necessity of these substances for the maintenance of health and normal growth. Vitamin B complex plays an important role in this respect. At present it is postulated that at least seven factors necessary for animal existence are found in B complex. There are too many controversies as to the effects and needs of all these separate factors. Vitamin B_1, known as the antineuritic vitamin, and vitamin B_2, or G, have been isolated and are available in pure form.

VITAMIN B_1

It has been definitely shown that vitamin B_1, or B, is of value in correcting and preventing anorexia of dietary origin, in securing optimal growth of infants and children, in curing and preventing beriberi and in treating some conditions in which difficulty is encountered in utilizing ordinary foods in the usual way, such as pernicious vomiting of pregnancy. It is claimed that vitamin B_1 has an influence on intestinal motility, and it appears to be in some way concerned in the metabolism of carbohydrates in the body. There is definite evidence that several forms of neuritis in man have vitamin B_1 deficiency as the main etiologic factor or as an important contributory factor.

It has been shown that a sufficient amount of alcohol destroys the proteolytic activity of certain gastro-intestinal enzymes in vitro and in vivo. It is suggested that alcoholic polyneuritis or deficiency disease may be caused, in part at least, by faulty digestion and assimilation of food resulting from the destruction of digestive enzymes by large quantities of alcohol taken over a considerable period of time.

In 1933 I [17] reported the cure of toxic amblyopia due to alcohol and tobacco in patients who were allowed to smoke and drink a moderate amount of alcohol when they were treated with a diet high in vitamins.

17. Yudkin, A. M.: Ocular Disturbances Produced in Experimental Animals by Dietary Changes: Clinical Implications, J. A. M. A. **101**:921 (Sept. 16) 1933.

Similar results have since been reported by Carroll,[18] who placed his patients under supervision in a hospital and observed cures in those in whom the optic nerve was not atrophied.

There are many instances of the cure of toxic amblyopia on removal of the offending drugs and the improvement of the general nutrition, but at no time has the condition improved so rapidly as when high vitamin therapy was employed. It is apparent that toxic amblyopia may be associated with a deficiency disease, for in the light of what has been said it appears that alcohol taken over a long period destroys digestive enzymes and thus prevents the proper digestion and assimilation of food.

VITAMIN B_2

According to Sherman,[19] vitamin B_2, or G, is essential to growth and to normal nutrition at all ages. When the food is poor in vitamin B_2 for any considerable length of time, digestive disturbances, nervous depression (different from the symmetrical polyneuritis of vitamin B_1 deficiency), general weakness and deterioration of tone and an unhealthy or unthrifty condition of the skin are likely to develop. The incidence of infectious disease seems likely to be increased, vitality diminished, life shortened and the prime of life curtailed by the early onset of senility. Evidence from several laboratories indicates that the heat-stable vitamin of the B complex, recognized as B_2, or G, consists of two factors: lactoflavin and B_6 (antidermatitic). B_2, or flavin (d-riboflavin), is important to the ophthalmologist, for in 1931 Day, Langston and O'Brien[20] described the consistent appearance of cataract in rats as a result of a deficiency of vitamin B_2, or G. This observation was corroborated by me[17] and by others when the instruction of preparing the diet and feeding it was carried out according to the directions given by Day and others. Recently Day and his collaborators[21] demonstrated that cataracts did not develop in rats on a diet deficient in vitamin B_2, or G, if from 60 to 90 micrograms of pure lactoflavin was added to the diet weekly. Lactoflavin is of particular interest because of the existence of a flavin enzyme—a dehydrogenase. This compound appears to be a combination of flavin and other radicals and is the first example of an enzyme having a vitamin as an integral part. Vitamin B_2 may also be

18. Carroll, F. D.: Analysis of Fifty-Five Cases of Tobacco-Alcohol Amblyopia, Arch. Ophth. **14**:421 (Sept.) 1935.

19. Sherman, H. C.: Chemistry of Food and Nutrition, New York, The Macmillan Company, 1937.

20. Day, Paul L.; Langston, W. C., and O'Brien, C. S.: Cataract and Other Ocular Changes in Vitamin G Deficiency, Am. J. Ophth. **14**:1005, 1931.

21. Day, Paul L.; Darby, W. J., and Langston, W. C.: The Identity of Flavin with the Cataract-Preventive Factor, J. Nutrition **13**:389, 1937.

present in the lens, for Fischer [22] in 1934 extracted a water-soluble green-yellow fluorescent substance which he believed to be lactoflavin. It is possible that more information concerning its presence or absence in the lens will be forthcoming. It is obvious that vitamin B_2 must play some role in the development of the lens and in protection against cataract in the albino rat.

VITAMIN C

From the experimental work and the clinical observations made with the products of nature containing vitamin C, it became apparent that vitamin C not only gave protection from scurvy but had many other important functions. It is true that there are many other disturbances which a low intake of vitamin C produces. Outstanding among the pathologic conditions is a weakening of blood capillaries. The defect in the capillaries in vitamin C subnutrition was shown by Wolbach and Howe [23] (1926) to result from a failure of the endothelial cells to form the intercellular cement substance, and they suggested that this failure extends to the connective tissue in other parts of the body. Hemorrhage due to the abnormal capillary fragility plays an important part in subclinical vitamin C deficiency. Eddy and Dalldorf [24] reported that weakened capillaries (capillary fragility) could be demonstrated in from 35 to 66 per cent of the children received from the economically poorer homes into the Grassland Hospital, Westchester County, N. Y. Most of these children responded promptly to treatment with antiscorbutic foods, thus showing that the capillary fragility here observed was mainly due to the shortage of vitamin C in the previous diet. It was shown that intravenous administration of vitamin C decreases the coagulation time of the blood. Beneficial results of intravenous and oral administration of vitamin C in cases of hemorrhagic diathesis, hematuria, purpura and essential thrombopenia have been reported by several authors. As yet no correlation has been demonstrated between capillary resistance and the concentration of vitamin C in the blood plasma or the urinary excretion of the vitamin. The estimations of vitamin C in the urine and of capillary fragility are utilized for determining the status of the vitamin C content of the body. Unfortunately, there seem to be individual variations in the urinary excretion of vitamin C, so that the test is not accurate, and capillary fragility may be due to other conditions.

It has been my privilege to treat many patients with hemorrhagic retinitis of various types with cevitamic acid. The treatment seemed

22. Fischer, F. P.: Die reduzierenden Substanzen der Linse, Arch. f. Augenh. **108**:527, 1934; Die fluorescierenden Substanzen der Linse, ibid. **108**:544, 1934.

23. Wolbach, S. B., and Howe, P. R.: Intercellular Substances in Experimental Scorbutus, Arch. Path. **1**:1 (Jan.) 1926.

24. Eddy, W. H., and Dalldorf, Gilbert: The Avitaminosis, Baltimore, Williams & Wilkins Company, 1937.

to shorten the time ordinarily required for the repair of this type of lesion. It was noted, however, that synthetic vitamin C does not have the same effect on retinal and vitreous hemorrhages as lemon juice. Lemon juice seems to clear the extravasation of blood and edema much more rapidly. I quite agree with Rusznyäk and Szent-Györgyi [25] that there is something else in lemon juice that is effective in breaking down and aiding in the absorption of hemorrhages of the vitreous, choroid and retina. It is my policy to prescribe for this type of ocular disturbance the juice of at least four or five lemons daily. May I suggest that a more careful and painstaking investigation be made with lemon juice and vitamin C therapy in ocular conditions of this nature? May I warn the investigator, however, that vitamin C is frequently not absorbed readily and that the source of the vitamin is variable?

Vitamin C is also considered of fundamental importance in tissue respiration, acting as a "hydrogen transport agent" between organic metabolites and, indirectly, molecular oxygen. It is discovered that crystalline lens and aqueous humor have a high reducing value toward the dye 2,6-dichlorphenolindophenol. In order to prove that the reducing substance concerned was cevitamic acid, the antiscorbutic activity of lens and aqueous was tested biologically; that is, lens tissue and aqueous humor were fed to guinea pigs which had been kept on a diet free from vitamin C until loss of weight occurred and symptoms of scurvy appeared; the vitamin C content of the tissue was estimated by the dose required to produce recovery. It was observed that the antiscorbutic potency of lens and aqueous as tested by the biologic method was much less than that indicated by the chemical estimation. The investigators concluded that this lack of agreement between the biologic and the chemical methods of estimation was due not to a lack of vitamin C in the tissue but to the presence of a toxic substance. Nothing is known with certainty regarding the function of vitamin C in the metabolism of the lens. It has been suggested that cevitamic acid and glutathione may be two linked factors in one system of oxidation in animal cells. A number of investigators have recently shown that various enzyme systems are activated or inhibited by vitamin C, but nothing is known concerning its relation to enzyme systems in the lens. The center of the lens, which respires less than the outer layers, contains less vitamin C.

The relation of vitamin C to the formation of cataract is problematic. It appears highly probable that this substance has some essential function in the metabolism of the normal lens, and if this is so, any condition which would lead to its withdrawal might be expected to induce degenerative changes. However, cataract is not associated clinically with

25. Rusznyäk, S., and Szent-Györgyi: Vitamin P: Flavonols as Vitamins, Nature, London **138**:27, 1936.

scurvy in human beings, nor is its occurrence recorded in experimental animals on scorbutic diets.

The observations recorded here are all fairly recent and many of them require confirmation. It is evident that more information as to the function of vitamin C in the normal lens is required before any theory as to its relation to the formation of cataract can be elaborated. Nevertheless, it has been my policy in recent years to give any adult patient showing early lenticular changes or tumescence associated with refractive changes the following therapeutic advice: Regulate the daily diet so that the proper amount of calories is obtained. Do not rely on any fanatic diet for sustenance. Before breakfast and dinner take the juice of at least one lemon, either as a lemonade or in some other non-alcoholic combination. Take a teaspoonful of potent brewers' yeast powder or some other equivalent vitamin B complex twice daily before meals. If there is a cardiovascular condition present, see that the system is compensating. The latter disturbance may require the aid of an internist. Up to the present time I am satisfied that when no permanent changes are present in the lens in the form of opacities it is possible to repair early damage in the lens by the aforementioned therapy.

To what extent vitamin D and E are factors in the maintenance of normal ocular function or in the repair of pathologic ocular conditions is impossible to say at present. Several ocular disturbances, particularly the formation of lenticular opacities, have been attributed to lack of vitamin D in the diet. It is my impression that when considerable vitamin D in the form of viosterol and other similar products is given to children and supplemented by exposure to the sun or to artificial sun rays, a distressing granular conjunctivitis is produced. Further investigation of these and other vitamins may change the present concept of their requirements in ophthalmology.

CONCLUSION

It is apparent from analyzing laboratory statistics and clinical observations that the vitamins play a definite role in the therapy of ocular diseases. Vitamin A seems to be useful in the treatment of corneal inflammations and chorioretinal disturbances. The normal function of the rods and cones of the retina are enhanced by the use of vitamin A. Vitamin B complex is useful in the treatment of toxic amblyopia, particularly that produced by the use of alcohol and tobacco. In patients in whom focal infection is thought to be the underlying cause of the ocular inflammation, a combination of vitamin A and B complex is helpful. Vitamin C has been used for extravasation of blood in the vitreous, choroid and retina, but I find that lemon juice is more beneficial. Lemon juice is helpful in ocular disturbances in which the lesion may be due to improper vascular compensation. At the present time I find no definite use in ophthalmology for vitamins D and E.

RESULTS OF STELLECTOMY IN THE TREATMENT OF PIGMENTARY RETINITIS

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In September 1933 I presented, in collaboration with Drs. Malbrán and Balza, before the Society of Ophthalmology of Buenos Aires [1] the results that we had obtained for the first five patients with pigmentary retinitis treated by stellectomy; later, in 1934, in another publication,[2] we reported on a total of twelve patients with pigmentary retinitis treated by this method (the first five being included) and summed up the effects of the operation.

The time that has gone by since the operation in these cases, the favorable change in many of these patients and the undoubted benefits following this operation in additional cases have encouraged us to dwell once again on the subject so as to draw the attention of specialists to the value of stellectomy in the treatment of pigmentary retinitis.

The indifference and doubts of ophthalmologists in regard to the practical value of operations on the sympathetic nervous system in pigmentary retinitis are due to unacquaintance with the etiology of the malady, its severity and the scarcity and inefficiency of the therapeutic resources available today. But facts in medicine are as powerful as laws.

It may be said that there is some quackery in suggesting sympathectomy for pigmentary retinitis, but it is evident that patients with this condition who have been operated on by this method have shown real improvement, and these results, whether based on quackery or on scientific principle, are eloquent.

In late years the circle interested in pigmentary retinitis has been agitated by works that have published the results of operations on the sympathetic nervous system for this disorder and reported cases in which unquestionable success was obtained with this procedure in spite of the irregularity of its effects.

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1. Caeiro, J. A.; Malbrán, and Balza, J.: Tratamiento de la retinitis pigmentaria por la resección del simpático cervico-torácico, Rev. Asoc. méd. argent. **47**:3403, 1933.

2. Caeiro, J. A.; Malbrán, and Balza: Resultados obtenidos en el tratamiento quirurgico de la retinitis pigmentaria por la extirpación del ganglio estelar, Prensa méd. argent. **21**:2245 (Nov. 28) 1934.

In Buenos Aires my associates and I have operated on several patients for whom stellectomy has given good results, and we have been pleased to see some of our ideas reproduced in foreign works.

Until our report was published the results in a few cases of Meighan,[3] Campbell [4] and Royle [5] gave glimpses of the possibilities of resection of the sympathetic chain.

In an emphatic manner we have praised stellectomy by "low resection," as the Americans call it, because we consider it the most complete and effective operation in regard to the anatomicophysiologic basis, and its results have not disappointed us.

Recently Drs. Villegas and Pasman operated on some patients with pigmentary retinitis (these cases have not been published), using stellectomy performed according to my technic, and they obtained in some of these cases results that are flattering.

In 1934 and 1935 important contributions on this subject were published by MacDonald and Mackenzie,[6] Gifford and de Takáts,[7] Walsh and Sloan,[8] Kerr [9] and Greene [10] in North America.

Before the Academy of Surgery of Paris in 1936,[11] in reporting on work on surgical intervention on the sympathetic nervous system carried on with Fontaine, Leriche drew attention to the benefits obtained in a patient with pigmentary retinitis who was operated on by this method.

To sum up the results obtained, all these authors, in spite of the caution that every new treatment inspires, advise research in order to arrive at more definite conclusions and suggestions regarding this procedure in view of the favorable changes seen in some patients operated on by this method.

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3. Meighan, S. S.: Treatment of Retinitis Pigmentosa by Sympathectomy, Tr. Ophth. Soc. U. Kingdom **51**:124, 1931.

4. Campbell, G.: Sympathectomy in a Case of Retinitis Pigmentosa, Canad. M. A. J. **26**:674 (June) 1932.

5. Royle, N. D.: Treatment of Blindness Associated with Retinitis Pigmentosa, M. J. Australia **2**:111 (July 23) 1932.

6. MacDonald, A. E., and Mackenzie, K. G.: Sympathectomy for Retinitis Pigmentosa, Arch. Ophth. **13**:362 (March) 1935.

7. Gifford, S. R., and de Takáts, Géza: Cervical Sympathectomy in Retinitis Pigmentosa: Preliminary Report on Results, Arch. Ophth. **14**:441 (Sept.) 1935.

8. Walsh, Frank B., and Sloan, Louise L.: Results of Cervical Sympathectomy in Pigmentary Degeneration of the Retina, Arch. Ophth. **14**:699 (Nov.) 1935.

9. Kerr, Harry: Surgical Treatment of Retinitis Pigmentosa, Am. J. Surg. **28**:364, 1935.

10. Greene, Louis: Personal communication to the authors, mentioned by Walsh and Sloan,[8] p. 713.

11. Leriche, R., and Fontaine, R.: Résultats généraux de 1,256 sympathectomies, Mém. Acad. de chir. **62**:877 (June 10) 1936.

ETIOLOGIC BASIS FOR SURGICAL INTERVENTION ON THE SYMPATHETIC SYSTEM

The etiology of pigmentary retinitis has not yet been determined exactly. For some time this disorder was considered to be due to a condition of the capillaries.

Subsequently Collins, although admitting the influence of the vascular alteration, stated that he considered this to be secondary, the original phase of the disease being atrophy of the neuro-epithelium.

The modern anatomicopathologic studies of Verhoeff [12] have confirmed Collins' ideas. Verhoeff considered the atrophy of the neuro-epithelium, particularly the layer of the rods, as a consequence of hepatic disturbance, the fundamental origin of the malady.

Vascular therapeutics were little by little abandoned, and treatment was directed in accordance with these new ideas, with the suggestion of hepatic opotherapy, the administration of insulin and vitamins and other treatment.

Nevertheless the results obtained with the reaction following sympathectomy and its influence in bringing about a better retinal circulation brought up again the vascular theory.

Suppression of the sympathetic innervation, by eliminating the constrictor action of the blood vessels, allows a greater affluence of blood through permanently paralytic vessels and expansion in all the vascular circuits of the eye (choroidal and retinal).

Some authors, such as Goltz and Freusberg,[13] have affirmed that some time after sympathectomy, the capillary functions are restored to their former status. These ideas at present cannot be admitted when ganglionic sympathectomy is performed, for experience has proved that with this operation the phenomena of vascular expansion that are attained are persistent (Diez [14]) or, at least, last a long time.

When Royle and Meighan performed cervical sympathectomy for pigmentary retinitis their intention was to modify the ocular circulatory condition in order to obtain better nourishment of the retina.

I believe that the suppression of the sympathetic innervation creates new conditions of life in the tissues, producing modifications of their nourishment and trophic state. These phenomena, which can be seen in all the tissues of the organism, are more pronounced the greater the cellular difference is.

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12. Verhoeff, F. H.: Microscopic Observations in a Case of Retinitis Pigmentosa, Arch. Ophth. **5**:392 (March) 1931.

13. Goltz, F. R., and Freusberg, A.: Gefässerweiternde Nerven, Arch. f. d. ges. Physiol. **9**:179, 1874.

14. Diez, J.: La tromboangeítis obliterante, Buenos Aires, El Ateneo, 1934.

The retina, the cells of which are ultradifferent, owing to its nature and functions, is more sensitive to any trophic or nutritive change than other parts.

Ganglionic sympathectomy in modifying fundamentally the trophism of the neuro-epithelial cells creates new protoplasmatic activity, stimulating a better cellular metabolism. The permanent vascular expansion and the greater affluence of blood change substantially the nutritive conditions of the cells, which awaken to new activity.

ELECTION OF PLACE AND SIDE FOR RESECTION OF THE SYMPATHETIC CHAIN

The methods of intervention suggested for suppression of the sympathetic innervation of the eye are: (1) resection of the superior cervical ganglion (Meighan [3]), (2) sympathectomy performed on the carotid ganglion (Magitot and Desvignes [15]) and (3) resection of the cervicothoracic sympathetic ganglion, or stellectomy (Royle, Frazier and Caeiro).

These three methods of intervention, apparently similar, differ fundamentally in the intensity and duration of the effect.

Place Chosen for Operation.—Present day knowledge shows that when the sympathetic paths to the head, neck and upper limb separate from the tractus intermediolateralis they concentrate in the cervicothoracic, or stellate, ganglion, the result of fusion of the third cervical ganglion with the first thoracic ganglion. From this point the sympathetic fillets go toward the head in three ways (1) by way of the carotid chain formed by the plexus of both carotid nerves, the internal and the external; (2) by way of the sympathetic trunk and its cervical, middle and upper ganglions, which end in the cavernous plexus, and (3) by way of the spinal nerve that runs in the direction of the spinal artery, ending in Willis' circle, in the arteries of the rear part of the brain and in the branches of the optostriate nucleus.

According to the diagram (fig. 1), it will be seen that sympathectomy performed on the carotid ganglion, as well as resection of the upper cervical ganglion, in comparison with stellectomy must be considered as segmentary sympathectomy, and its value is less in reference to the elimination of the number of sympathetic fibers.

Sympathectomy on the superior cervical ganglion performed in association with sympathectomy on the carotid ganglion does not destroy totally the sympathetic innervation of the eye.

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15. Magitot, A., and Desvignes: Les effets oculaires de la décortication carotidienne (sympathectomie artérielle) chez l'homme, Bull. Soc. d'opht. de Paris, March 1934, p. 143.

Besides, on another occasion we have shown that sympathectomy of the carotid ganglion, by provoking incomplete destruction of the sympathetic fibers, can produce the opposite of the desired effect, that is, excitation of the sympathetic function, which would naturally produce constriction of the ocular vessels, an effect totally different from the one sought.

The stellectomy praised by Royle and by us, which is classified by Americans as a "low" operation, is anatomically and functionally the

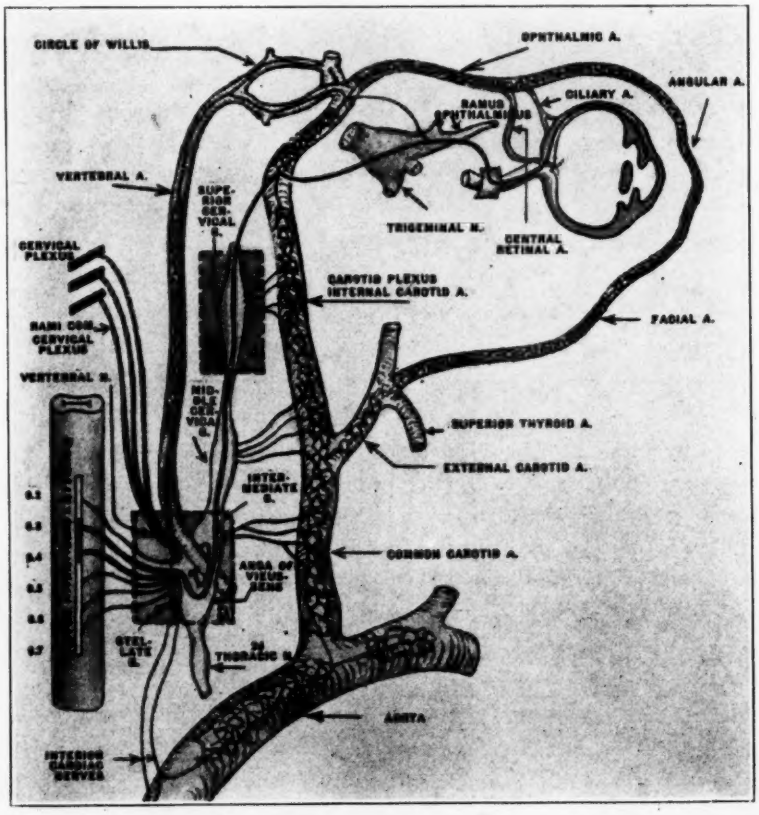

Fig. 1.—Sketch of the sympathic pathways of the eye. *A* illustrates segmentary sympathectomy, or resection of the upper cervical ganglion, and *B*, stellectomy, or resection of the cervicothoracic ganglion, a more complete operation.

exact intervention to attain the maximum of effect by total destruction (within the present day knowledge) of the sympathetic innervation of half of the head. I place much value on, and systematically perform, resection of the spinal nerves, which in some cases of thick intermediary ganglions that may be confused with the mass of the stellate ganglion may escape cutting (section).[16]

16. Caeiro, J. A.: La estelectomía, Semana méd. **1**:557 (Feb. 21) 1935.

I perform section of the connection of the stellate ganglion and of the second dorsal nerve a little high in order to preserve as much as possible the greater part of the rami communicantes that go toward the last cords of the brachial flexor muscle—the sixth, seventh and eighth—and the first dorsal nerve, as these are not necessary for the purpose, also avoiding useless sacrifice of part of the sympathetic innervation of the upper limb.

Stellectomy is an operation that must be closely controlled by the surgeon to avoid failure due to poorly performed or incomplete extirpation. Since it is a purely anatomic operation, search for the stellate ganglion may be easy in some cases, and may be very complicated in others, more so in those cases in which fusion of the inferior cervical ganglion with the first thoracic ganglion is incomplete. A surgeon of little experience may easily make the mistake of extirpating what looks like the stellar ganglion and what really belongs to that bulkiness known as the intermediate ganglion. Extraction of this ganglion will be an incomplete operation, for the stellate ganglion and the spinal nerve will not have been touched. Of course the failure in these cases cannot be considered as due to the method applied but must be thought of as due to the defective technic employed.

Claude-Bernard-Horner's syndrome, which unavoidably must occur, no matter how high the sympathetic trunk and Vieussen's ansa, that is the way that the iridospinal fillets take to reach the eye (fig. 1), are sectioned, must not be taken as a sign that the stellectomy and the section of the spinal nerve have been performed correctly or as indicating a good prognosis, as some mistakenly interpret it.

Incomplete resection in which the lower part of the stellate ganglion and the beginning of the spinal nerve are left will be accompanied by Claude-Bernard-Horner's syndrome, but for the desired purposes it will be a poorly performed and ineffective operation.

On the other hand, the absence of Claude-Bernard-Horner's syndrome after the operation means a poorly carried out operation, as the sympathetic chain has not been sectioned.

Therefore we have already remarked that this operation must be strictly controlled by the surgeon in order to draw useful deductions from the results, unsuccessful as well as successful.

Side on Which Operation Is Performed.—There is no special factor that determines definitely the side to be chosen for the intervention.

Nevertheless, if one considers the relationship, with the effect desired and the modifications that one strives to attain when one changes the trophic state of the neuro-epithelium and modifies the state of the circulation, choroidal and retinal, naturally the side chosen must be that on which the patient shows keener and better sight.

The retina with better function will show a smaller degree of atrophy than the other retina, and of course the effects sought will be more intense in a retina with good function than in a retina that has lost all function. In any case the effects are felt on the opposite side, though in a minor degree.

The results that my associates and I have obtained following this idea appear to us to be safe, as we place ourselves in a position to work on the retina that has the fewer functional and anatomic injuries.

OCULAR MODIFICATIONS

The ocular modifications obtained were: first, those dependent on the suppression of the sympathetic innervation; second, those due to the changes in the optic function, and, third, those due to the contralateral ocular modifications.

1. In all our patients stellectomy has immediately called forth Claude-Bernard-Horner's syndrome. The regular appearance of this syndrome is due to the severing of the fillets going to Müller's muscle and to the elevator muscle of the upper eyelid. The absence of this syndrome after stellectomy means that the operation has been performed incorrectly.

Enophthalmos and miosis vary according to the patient. The vascular expansion that takes place a few minutes after the operation becomes distinct after eight or ten hours, reaching its full intensity within twenty-four or thirty hours. It lasts three or four days, though in some cases we have noticed that it was intense during a week and afterward gradually diminished until it disappeared within fifteen or twenty days. We have noticed an interesting fact that may be taken as of prognostic value: The greater the conjunctival vascular expansion, the more favorable will the outcome be in most cases.

2. The changes in the optic function are seen in two fundamental phenomena: (*a*) modifications in visual keenness and (*b*) modifications in the visual field.

a. Generally, visual keenness, except in the patients in whom the injuries were very advanced and who were operated on in order to prove how much it could be modified (persons who were almost blind), has shown an increase in most cases. Sharper sight has appeared in the eye on the side operated on, and we believe that this was due to performing the operation on the side on which sight was keener.

This increase in visual keenness has been as variable in intensity as the time of its appearance. It has generally appeared within the first twelve or twenty-four hours. The patients noticed it especially at night, when they remarked a greater brightness in the electric light bulb and an absence of the mistiness that they were in the habit of seeing.

In some cases the visual keenness remained stationary and later increased slowly but progressively within the first month. In other cases it reached the maximum a few days after the operation, vision being doubled or trebled. Only one patient, after three months, lost much of what he had gained, but nevertheless his vision remained better than it had been before the operation.

In some cases, though in a very irregular way, improvement in visual keenness, though variable, appeared on the side opposite the one on which the operation was performed.

It has been proved that the greater the congestive and heat reaction of an eye, the quicker the increase in visual intensity. This fact shows clearly the beneficial action of a better retinal circulation.

One can practically consider that increase of visual keenness, in spite of the fact that it takes place within variable limits, is almost constantly seen after the operation when this is performed on a patient who has not reached the point of seeing only bulky objects.

b. The visual field also undergoes modifications, although they do not occur with as much regularity as increase in visual keenness. The field nearly always shows an increase of from 10 to 20 degrees, and in only one case (observation 7 of Caeiro, Malbrán and Balza [2]) was a considerable increase achieved.

According to the way in which we think stellectomy acts, through modification of the circulatory condition and cellular trophism, improvement of the visual field is made possible because of the better cellular nourishment. A phenomenon takes place similar to that observed in Volkmann's ischemia in certain types of thrombo-angeitis of the lower limbs and even in angina pectoris, in which, provided the anatomic injury of the cells has not led to final atrophy, operation on the sympathetic chain can modify the condition and bring about improvement of these ailments.

When cellular disturbances are definite, operation on the sympathetic nervous system is absolutely useless.

This fact of great importance must be taken into consideration in order not to attach the blame for failure to sympathectomy when this operation has been suggested too late.

The retina cannot escape the effects of general pathologic changes, and whether these effects are degeneration of the neuro-epithelium, narrowing of the light reflex of the retinal vessels through proliferation of the conjunctival tissue or disturbances of vascular type, operation on the sympathetic chain may be useful when practiced early, allowing an increase in the circulation of the retina. This will produce a favorable reaction that will show in a better metabolism and cellular activity and consequently better function, that is to say, better sight.

If, on the other hand, the operation is performed too late, little or nothing can be achieved, for the retina is anatomically and functionally lost.

3. *Contralateral Ocular Modifications.*—My associates and I have remarked the beneficial influence of the operation on the function of the retina of the opposite side in four cases and its manifestations as regards, in the first place, visual keenness and, in the second place, the visual field.

The phenomena observed took place immediately after the operation or within fifteen or twenty days or even later. The patients drew our attention to this improvement, and in the beginning we thought it might be based on imagination, but later, when the visual keenness was controlled, it was seen that vision really increases.

What mechanism can explain these facts? To leave the ocular sphere and to enlarge on the phenomena that are seen immediately after the operation on the sympathetic system, frequently one sees effects that appear to be due to suppression of the sympathetic innervation on the side opposite to the one on which the operation has been performed. We have been able to prove this, and Royle [5] has also drawn attention to the effects of severing a branch in spastic paralysis. On one occasion, in a patient on whom we performed sympathectomy of the femoral nerve, we noticed increase of oscillations and temperature in the limb on which the operation was not performed. We also have had a chance to remark phenomena of paralytic vascular expansion after stellectomy on the side on which the operation was not performed.

The crossed sympathetic innervation and the anastomosis between the hips are a feasible explanation for these phenomena, so far as the lumbar portion of the sympathetic system is concerned, but in the cervical chain, within the present day knowledge, there is independence between the right side and the left side (Caeiro [17]).

We attach importance to the appearance of these phenomena after resection of the spinal nerve, which, when it is done in a complete manner, can destroy part or all of the plexus that accompanies the trunk of the basilar artery and which can influence the paralytic vascular expansion of the choroidal circulation of the sound side through production of partial destruction, or maybe phenomena of functional inhibition also intervene.

The truth is that one cannot give a satisfactory explanation of these phenomena with the present day knowledge. Neither can the anatomic features of the sympathetic system that the knife discloses give such

17. Caeiro, J. A.: Resultados de la estelectomía en la parálisis traumática del nervio facial, Bol. y trab. de la Soc. de cir. de Buenos Aires **19**:392 (June 26) 1935; Semana méd. **1**:572 (Feb. 20) 1936.

an explanation. Possibly they are phenomena of correlation of functional order due to an unknown anastomosis of which only the physiopathologic characteristics of this system, as one goes deeper into the study, will give a more definite explanation.

GENERAL MODIFICATIONS RESULTING FROM STELLECTOMY

The alterations that stellectomy produces by destroying extensive zones through which the peripheral portion of the sympathetic system is distributed cause disturbances that vary in intensity and in duration.

Vasomotor Alterations.—These have generally been temporal and have been associated in a greater degree with changes in the weather, with sudoriferous disturbances and with pilomotor phenomena. These changes have been variable, and some have been more emphasized than others, but generally they have soon disappeared.

With more regularity violent headaches of congestive type have occurred, sometimes persisting during the first few days after the operation; in only one patient have we seen them reappear from time to time and lasting a very short while.

Pain.—Some American authors have stressed the occurrence of pain. This is of two kinds: that which starts in the precordial region and crosses to the level to the spinal edge of the omoplate and that which occurs in the upper limb. The first kind of pain usually disappears quickly. On the other hand, pain in the upper limb occurred in the first cases in which my associates and I extended the resection almost to the second thoracic ganglion, in such a manner that the branch communications to the branchial plexus were completely destroyed; today we have partly modified our operation and have reduced it to extirpation of only the stellar ganglion and particularly to resection of the spinal nerve; this type of pain has hardly ever appeared with this procedure, as part of the sympathetic innervation of the upper nerve is not touched.

In the few cases in which there has been pain it has been temporal and has been confined to the deltoid region and the root of the shoulder. As to the pain in the neck and head, this corresponds more to the cephalea already mentioned.

Cardiac Disturbance.—This has been one of the most important objections made to stellectomy. In all our patients with a sound cardiovascular apparatus my associates and I have not remarked any alteration that could upset the normal cardiac function, even in a case in which stellectomy was performed bilaterally. This has led us to believe that the objection is based more on theory than on fact, and it does not find confirmation in the results of the finest methods of investigation of the cardiac function. We have taken the precaution of making an

electrocardiogram for all our patients before and after the operation, and electrocardiograms have also been made later. None of these electrocardiograms has shown disturbances of waves or conduction, and the latter have kept within normal limits, even in the cases in which stellectomy was performed bilaterally.

These results are evident facts that we do not pretend to explain, but they have a certain importance, and we feel justified in believing, maybe somewhat empirically, that stellectomy practiced on patients who have the cardiac apparatus undamaged will cause little disturbance through the suppression of the sympathetic innervation.

RESULTS

The patients for whom operative results can be reported may be divided into two series: The first series consists of thirteen patients operated on without consideration of the degree of progress of the ailment. In some of the patients of this group the malady was so far advanced that they could practically be considered blind. The decision to operate was made expressly to see how stellectomy could modify the condition.

The results already published [2] are those for four patients who had a great increase in the visual field and in visual keenness, which still persists. One patient who obtained much improvement in visual keenness and in his field lost within six months almost all that he had gained; a slight improvement of visual keenness nevertheless persisted.

In four patients only a slight improvement in visual keenness was obtained, and lastly, in four others there was no change.

The good results in the first four patients can be considered well proved. One of these was operated on in March 1933. His improvement was very marked and allowed him to return to his work. This patient came back some time later and requested us to operate on the opposite side, in view of the good result of the first operation. He was operated on in 1936 and did not obtain more benefit than that already obtained through the first operation.[18]

The second series includes selected patients, that is, those in whom the malady was not very advanced or at least whose visual keenness permitted some degree of useful sight. All these cases are unpublished cases; the patients were operated on in 1936, with very satisfactory results.

REPORT OF CASES

CASE 1.—The patient was M. B. H., a married Argentine woman 30 years of age. Her father had died of heart disease at 67 years of age. Her mother was still alive and was in good health. The patient had seven brothers. One

18. Caeiro, J. A.: Retinitis pigmentaria, Bol. y trab. de la Soc. de cir. de Buenos Aires **19**:1131, 1935.

of them had pigmentary retinitis; the rest were well. The patient had had no abortions and had two healthy children. As far back as she could remember she had had pigmentary retinitis, with hemeralopia. She was well built, strong and in perfect physical health. An electrocardiogram was normal.

Visual keenness before operation was 1/3 for the left eye and 1/4 for the right. The visual field of the left eye, as determined with a 3 mm. test object at a

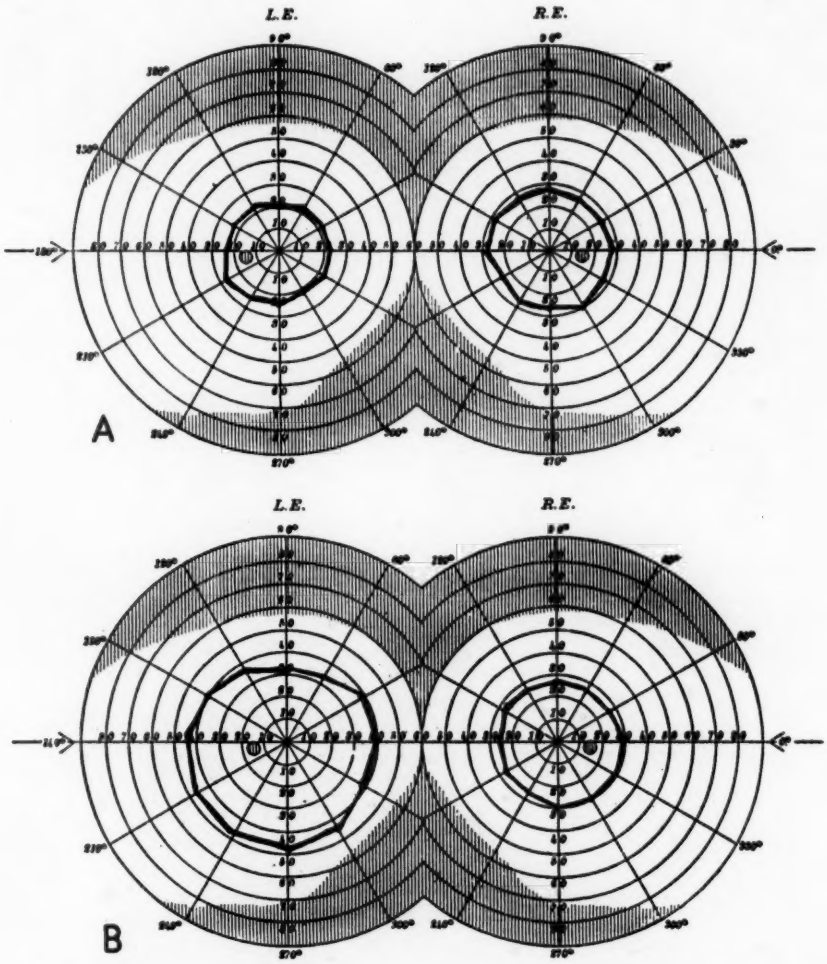

Fig. 2. (case 1).—*A*, fields before operation, on Aug. 16, 1936, as determined by a 3 mm. test object at 330 mm. Vision of the left eye was 1/3 and that of the right eye 1/4. *B*, fields after operation, on October 16, as determined by a 3 mm. test object at 330 mm. Vision of the left eye was 1/2 and that of the right eye 1/4.

distance of 330 mm., was contracted to 20 degrees and that of the right eye, as determined similarly, was contracted to 25 degrees.

Stellectomy was carried out on the left with our usual technic, in September 1936.

After the operation the condition was normal. On examination on Oct. 16, 1936, the visual keenness of the left eye (the eye on the side operated on) was 1/2, and the visual field, as determined with a 3 mm. test object at a distance of 330 mm., extended both nasally and temporally to 40 degrees.

This patient's progress was most satisfactory; the visual disturbances at night became less pronounced. When examined in January 1937 she still showed improvement. The electrocardiogram taken at this time showed nothing of import.

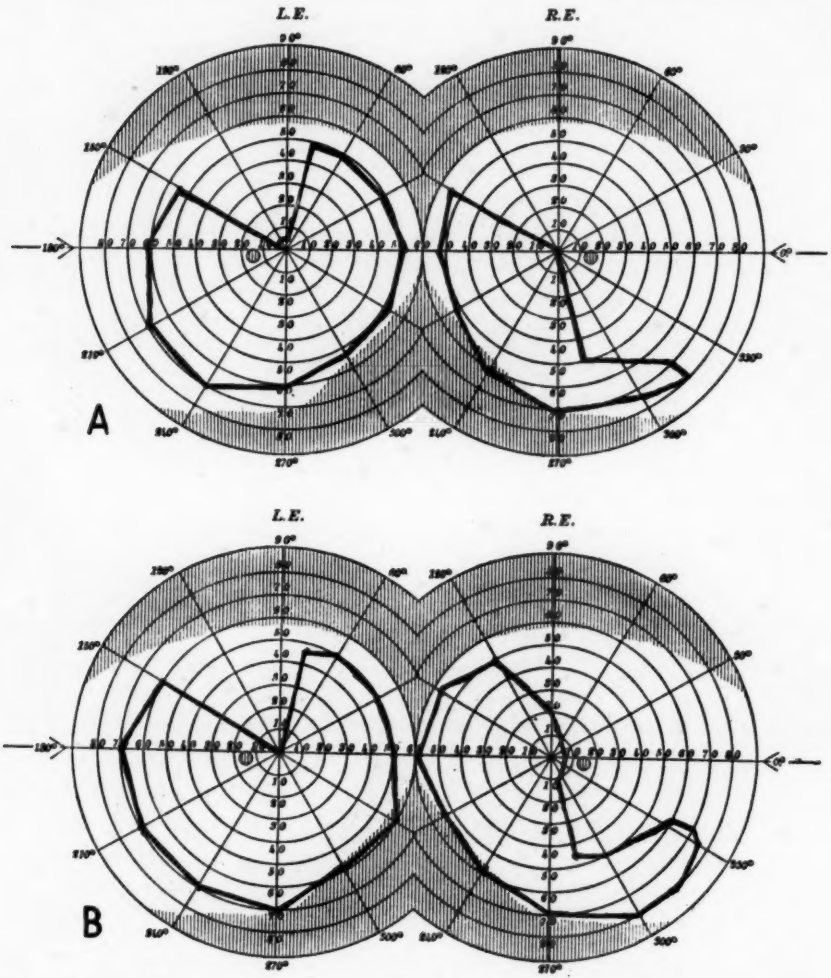

Fig. 3 (case 2).—*A*, fields before operation, on April 17, 1936, as determined by a 3 mm. test object at 330 mm. Vision of the left eye was 1/5 and that of the right eye 1/4. *B*, fields after operation, on October 26, as determined by a 3 mm. test object at 330 mm. Vision of the left eye was 1/3 and that of the right eye 1/2.

CASE 2.—L. G., a married Polish man 47 years of age, had hereditary and personal antecedents of no importance. No member of his family had suffered from pigmentary retinitis. His malady began two years previously with loss of

vision, particularly at night, with marked diminution lately. He could not get along alone and could hardly distinguish very large letters.

On April 17, 1936, examination of the visual field of the left eye with a 3 mm. test object at a distance of 330 mm. showed contraction to 50 degrees in the nasal fields and to 70 degrees on the temporal side.

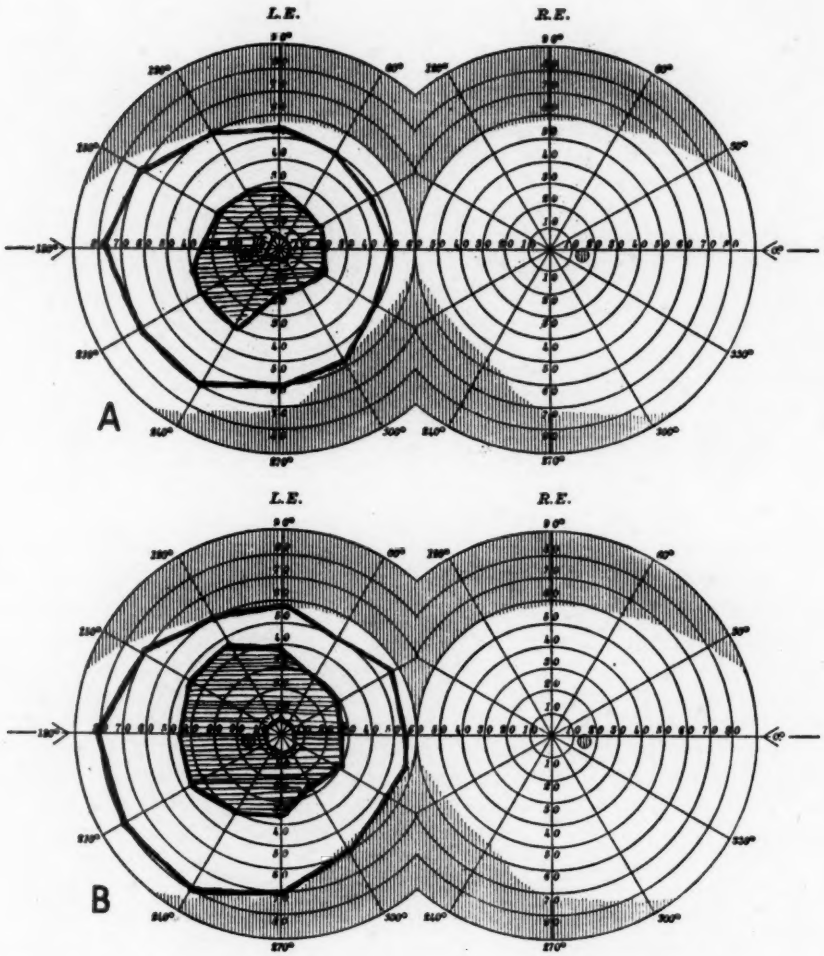

Fig. 4 (case 3).—*A*, fields before operation, on June 30, 1936, as determined by a 3 mm. test object at 330 mm. Vision of the left eye was 1/10 and that of the right eye 0. *B*, fields after operation, on October 27, as determined by a 3 mm. test object at a distance of 330 mm. Vision of the left eye was 1/6 and that of the right eye ability to distinguish bulky objects.

The visual keenness was 1/5. The visual field of the right eye, as determined with a 3 mm. test object at a distance of 330 mm., was contracted on the nasal side to 60 degrees, the extension on the temporal side being 0.

The visual keenness was 1/4. The electrocardiogram taken before the operation showed nothing important.

On May 5 stellectomy was carried out on the right. The immediate results were very good, visual keenness increasing so much as to allow the patient to read and to get along by himself. The electrocardiogram made after the operation was normal.

Examination of the visual keenness made in October showed vision of 1/3 for the left eye, and vision of 1/2 for the right. The visual field of the left eye

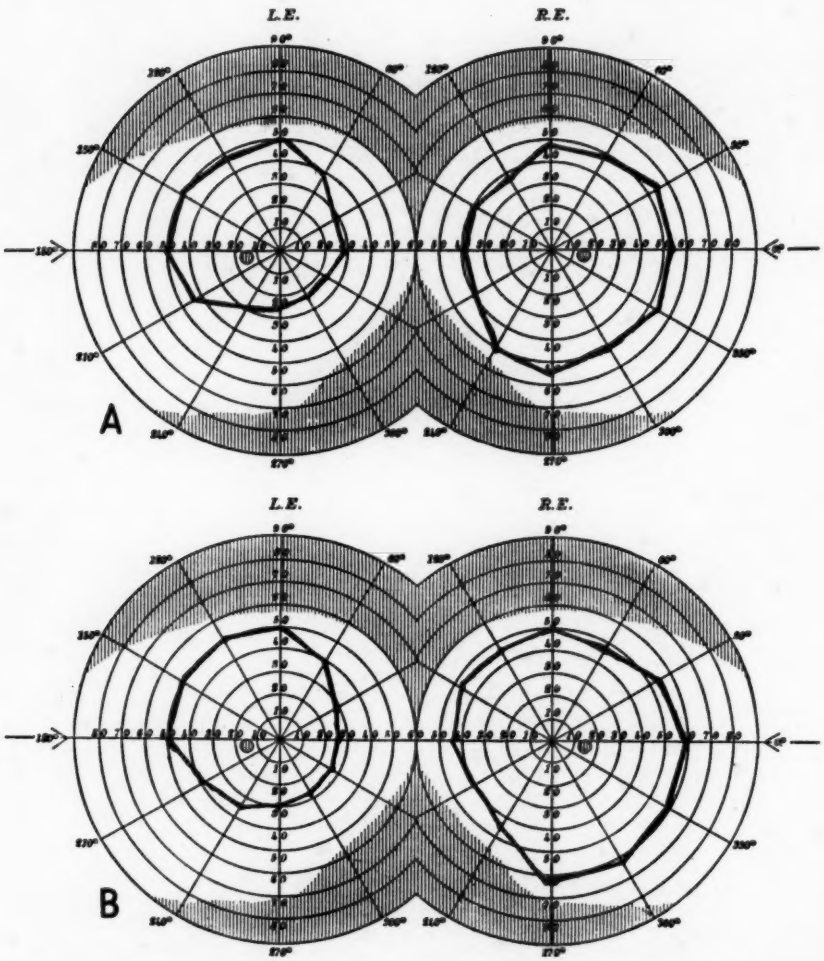

Fig. 5 (case 4).—*A*, fields before operation, on May 8, 1936, as determined by a 5 mm. test object at 330 mm. Vision of the left eye was perception of fingers at 3 meters and that of the right eye 3/10. *B*, fields after operation, on October 26, as determined by a 5 mm. test object at 330 mm. Vision of the left eye was perception of fingers at 3 meters and that of the right eye 1/2.

remained within the same limits as previously, but that of the right eye had increased in the temporal zone to 50 degrees.

In this patient the contralateral effects were amazing.

When examined on Jan. 5, 1937, he was found to be doing well, and he has returned to his work.

CASE 3.—The patient was J. W., a married Polish man 48 years of age. The father was dead, the cause of his death being unknown to the patient. The mother was alive and healthy. The patient had five brothers, all healthy, and had two healthy children. The patient had no ancestors who had had pigmentary retinitis. His malady began twenty years before; his sight had diminished gradually, and at the time of examination he could see only bulky objects and had been forced to abandon his work.

Ophthalmoscopic examination on June 30, 1936, showed visual keenness to be 0 in the right eye and 1/10 in the left. Determination of the visual field of the left eye with the use of a 3 mm. test object at 330 mm. showed a central area, with a peripheral zone extending between 25 and 55 degrees. An electrocardiogram made before the operation was normal.

On July 3 stellectomy was carried out on the left with the usual technic.

The next day the patients was able to distinguish the separation between letters, whereas before the could see only one continuous black line. Two days later he saw clearly the objects within reach of his hand and could read the posters on the wall. He was now able to walk alone.

Ophthalmoscopic examination on October 27 showed the visual keenness of the left eye to be 1/6 without correction and that of the right eye ability to distinguish bulky objects. The visual field had increased in the central and peripheral portions. An electrocardiogram was normal.

An examination was made again in January 1937. The improvement had persisted, allowing the patient to work again and get along alone.

CASE 4.—J. S., a single Italian man 37 years of age, had antecedents of no importance. There was no pigmentary retinitis in his family. Since his childhood he had been gradually losing his sight, and this loss had been marked in the previous ten years. There was hemeralopia. A month before examination his sight diminished rapidly.

Ophthalmologic examination on May 8, 1936, showed that the visual keenness of the left eye had not been modified, and the patient could see fingers 3 meters away. The visual field of the left eye, as determined with a 5 mm. test object at a distance of 330 mm., was contracted to 50 degrees on the temporal side and to 25 degrees on the nasal side. The visual field of the right eye, as determined similarly, was contracted to 50 degrees on the temporal side.

Operation was performed on the right on June 4, 1936; there were no immediate results.

The patient was examined again in October, when the ophthalmologic examination showed visual keenness of the right eye to be 1/2 with correction. The visual field of the left eye, as determined with a 5 mm. test object at a distance of 330 mm., was still contracted to 50 degrees on the temporal side.

The patient had improved very much. An electrocardiogram was normal.

Lately we have treated four more patients with this method, with good results, but owing to the short time that has elapsed since the operation we have not included the report of these cases here.

SOME NEWER DEVELOPMENTS IN PRECISION TYPE STEREOSCOPES

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Before describing some of the mechanical details of two new instruments to be presented here, it may be well to discuss briefly the reasons which prompted me to venture into creating and designing such devices as well as the principles favoring their uses.

The tendency of subjecting persons with supposed or actual muscle anomalies to orthoptic training solely on the basis of routine ocular study did not appear satisfactory, because one could not accurately or conveniently adjust the stereoscope to correspond to one's clinical findings. It seemed logical that an instrument designed to correct a binocular disturbance should also provide a definite means for gaging progress or improvement, which, in addition, might be used to determine both qualitatively and quantitatively the status of the eyes, so that the examiner could accurately decide whether the patient really required orthoptic training or just impressive ocular calisthenics. Hence, an optical instrument that is therapeutic would better meet critical standards if it could also be diagnostic or recording. In other words, a routine clinical study supplemented by an instrumental investigation should be the prerequisite to orthoptic training.

It seemed reasonable to understand first the normal binocular changes when one looked into a stereoscope; to be able to explain why certain separations of the split stereogram or the viewing distance proved comfortable and other positions induced diplopia. And so, before even constructing an experimental model, the various binocular phenomena induced by changing the viewing lenses and altering the lens separation, the viewing distance or the interstereogram separation were calculated on paper and subjected to experimental and clinical confirmation on normal as well as on abnormal subjects. These studies as well as the experimental model formed the basis of a contribution to the 1935 Scientific Exhibit of the American Medical Association and were more recently reported elsewhere.[1]

In short, this research revealed a few basic principles:

(*a*) The eyes cannot maintain the same pupillary separation with changes in the viewing distance.

1. Krimsky, Emanuel: The Stereoscope in Theory and Practice, Also a New Precision Type Stereoscope, Brit. J. Ophth. **21**:161-197 (April) 1937.

(*b*) As the eyes converge with accommodation, the interstereogram separation must be made correspondingly less in order to maintain easy fusion.

(*c*) A split stereogram which could be shifted to correspond to changing positions of the visual axes with changing accommodations is the only practical method for stereoscopic study.

(*d*) A stereogram with separation fixed for infinity viewing range becomes relatively divergent when brought nearer the eyes.

(*e*) The variable convergences of the eyes with changing accommodations can be accurately calculated and applied to stereoscopic investigation.

In order to be of practical value for the purpose described, the stereoscope should meet the following requirements:

(*a*) It should be calibrated in every respect so that the examiner can have absolute control in the selection of viewing lenses, lens separation, viewing distance or interstereogram separation.

(*b*) The instrument should be devised so that the examination will not be too time consuming.

(*c*) It should have psychologic appeal to both the examiner and the patient.

(*d*) It should be elastic in scope, not only as regards lateral ductions and phorias but as regards vertical and rotary ductions and phorias. (My instrument was even provided with a stereocampimetric attachment.)

(*e*) It should be a mechanically refined precision instrument that can yield reliable numerical readings.

(*f*) It should enable the examiner to observe readily the eyes of the patient without attracting the latter's attention.

(*g*) It should be simple to operate and require as few basic pictures or targets as possible to serve its purposes as a diagnostic as well as a therapeutic instrument.

My experimental model was further improved and modified to meet these requirements. In short, I aimed to improve the familiar Brewster stereoscope much as Howard, Pugh, Maddox and others have so well done with the more primitive Worth amblyoscope.

The value of a calibrated, or phorometric, stereoscope as a clinical instrument can be dealt with at length and will form the basis of a separate paper. A precision stereoscope enables the examiner to determine readily the divergence and the convergence status of the eyes both at infinity and at any desired range of accommodation and thus to diagnose and classify the type and degree of muscle anomaly. He can also determine the total range of fusion as well as of its component phases, adduction and abduction; the amount of phoria; the vertical and rotary

status as well as the horizontal status; the measurement of false projection, if any; the measurement of lateral as well as vertical deviations, and the progress at each sitting in terms of prismatic vergence.

While such an instrument could serve the purposes of a diagnostic as well as a training instrument, for the sake of economy and portability a junior, or companion, stereoscope was more recently developed, primarily as a home-training unit or one to be readily taken back and forth to the clinic. A separate description of each instrument may better explain the need for such an addition, and the two instruments will be described under the following headings: (1) senior model, or phorometric stereoscope (fig. 1), and (2) junior model, or companion stereoscope (fig. 2).

SENIOR MODEL, OR PHOROMETRIC STEREOSCOPE

The senior stereoscope (fig. 1) presents the following features:

(*a*) The viewing head consists of a simple, sturdy lenseless trial frame with compartments for two sets of trial lenses as well as multiple spring holders for the free insertion of supplementary square prisms. This frame may be readily adjusted to a lenticular separation that can be varied from 52 to 90 mm. The lens holders are calibrated to permit the insertion of cylinders at any desired axes. Thus, an ametropic correction may be added to the viewing lenses to determine the binocular status before glasses are prescribed.

(*b*) One of the connecting, double rods between the viewing boxes and the viewing head is marked off in centimeters and not in accommodation equivalents, so that the examiner may feel free to employ whatever strength lenses he chooses in the viewing head. The viewing distance may be readily changed from 12 to 40 cm. by the simple turning of a rack and pinion screw. This wide range permits flexibility in the selection of lenses for different requirements.

(*c*) The viewing boxes, or carriers, are designed to receive transparencies and solid objects as well as opaque prints. Whereas in the familiar stereoscope fixed stereograms are in the main relied on, in my instruments I employ split pictures which permit flexibility in movement and control in operation through the riding of both boxes simultaneously over a two way thread. This is accomplished by turning a handle which is placed conveniently within reach. The thread chosen is such that each complete turn of the handle moves each picture through a distance of 1 mm. Thus the operator is saved frequent inspections of the viewing scales.

(*d*) For purposes of precision, the position and lateral displacement of the split pictures or objects may be readily noted by means of an adjustable pointer which intersects two millimeter rules: a lower one

that is fixed and has the zero at its septal margin with the graduations increasing to the right and an upper slide rule that has its zero point at the center with graduations increasing from both sides of the zero. This rule moves with the movement of the boxes after its zero is set to the center or selective point of whatever size picture is employed.

In a precision stereoscope the exact separation of the pictures is important in relation to the known separation of the viewing lenses as

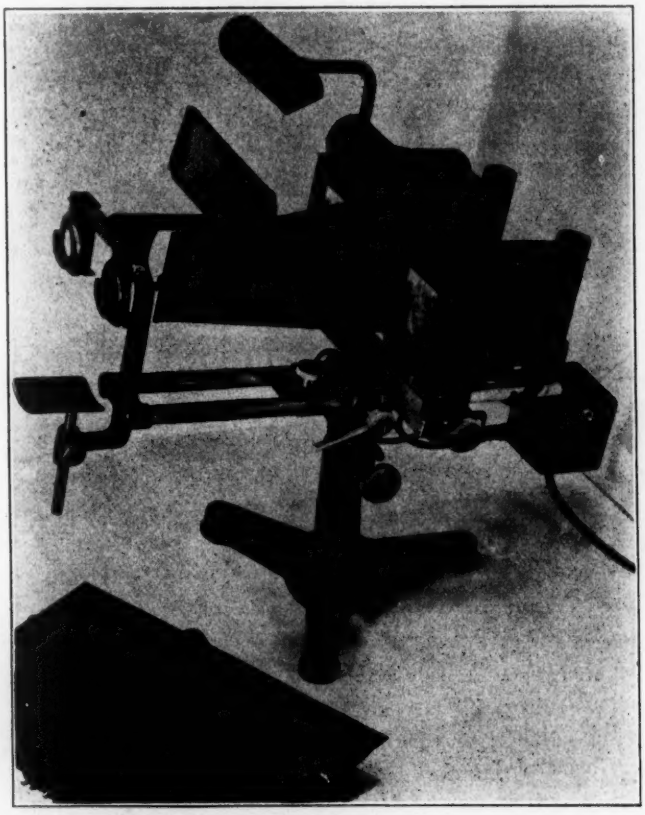

Fig. 1.—The senior, or phorometric, stereoscope (minus the adapters). This photograph is not retouched.

well as of the viewing distance. If the viewing lenses are set for infinity (their focal length), they would have to be separated from center to center by the same amount as the centers of the stereoscopic pictures in order to render the visual axes of both eyes parallel or to register in normally functioning eyes a single fused image for far vision. If, for example, the lenses are set at the 80 mm. separation (40 mm. from the midline), the center of the picture would likewise have to be set at 40 mm. on the lower fixed rule for this primary or parallel position. If

40 mm. on the fixed rule is selected as the starting point for operations, the pointer is set over the 40 and the center of the picture brought in line with it; the upper slide rule is then shifted with its zero between the 40 and the center of the picture. In this way, whatever displacement is made in the viewing boxes, either internal or external to the primary position for far vision, is determined by the displacement of the movable rule in relation to the adjustable pointer, which remains set over 40 mm. on the fixed rule. One may wish to determine the capacity of the eyes to maintain single binocular vision in the abduction phase or in the adduction phase. In either case the boxes are shifted until there is a break in the images, and then the reading is made on the slide rule in terms of millimeters. By means of a ready reference card this is readily translated into prism diopters and is recorded as the "breaking point." The boxes are then gradually brought back toward the primary position until a single binocular image is obtained, and this is likewise translated in terms of diopters as the "recovery point." And so one can graphically record for both the abduction and the adduction phase the respective "breaking points," which added together mean amplitude of fusion; one can also determine the "recovery points." The significance of such readings will be dealt with separately.

It is to be emphasized that the 40 mm. setting was merely arbitrary, and by means of simple tables the operator may at random vary the lenses, the lens separation or the viewing distance and readily make his translations. It is also to be emphasized that because eyes converge with accommodation the calculated primary positions or picture centers are not the same within infinity range as they would be at the focal length of the lenses. Here, too, the variable primary settings, with different ranges of accommodation, may be easily determined from such a table. And, finally, the element of interpupillary separation can be ignored for reasons discussed in a previous article.

For testing vertical or rotary phorias and ductions, suitable adapters may be inserted and manipulated to yield desired readings.

(*e*) The precision stereoscope offers the examiner and his patient a variety in the choice of stereoscopic material. Provision has been made by the means of suitable adapters for pictures of five different sizes: 45 by 45 mm., 60 by 60 mm., 75 by 75 mm. and 82 by 82 mm. (synoptophore slides) and in addition there is an adapter which holds both the Sattler and the Guibor split charts. The advantage of the smaller picture lies in its greater range of movement for both adduction and abduction. The larger picture is more appealing for its wider field of view. Moreover, the first three sizes were also chosen to correspond to the more popular sized stereoscopic cameras, so as to enable the examiner to make his own pictures if he so desires.

(*f*) Each viewing box has an independent lighting unit which operates both the lamp on the top of the box for opaque prints as well as the lamp within the box for transparencies. By a simple turn of the switch in the rear of the instrument, the light may be shifted to one or to the other. In this switch box are also independent rheostats to vary the intensity of illumination to the respective viewing boxes as well as independent flashers.

(*g*) One of the shortcomings in most examining or training instruments is that the examiner cannot see the patient's eyes follow the shifting of the pictures. This difficulty has been nicely overcome in my senior instrument by the incorporation of a tilted mirror, whereby inspection of the patient's eyes can be made without the patient being conscious of it. The mirror is conveniently set as to obviate unnecessary manipulations by the examiner, who faces both the mirror and the viewing boxes with its calibrations.

(*h*) The instrument has a collapsible telescopic double septum that is automatically adjustable to changing viewing distances. It consists of two opaque curtains which extend forward from respective rollers behind the corresponding viewing boxes. After hugging these boxes in such a manner as to guard against leakage of stray light when separated, the curtains continue forward to meet in front at the viewing frame, where they are attached. This septum may be readily released from its attachment when desired, so as to enable the examiner to insert a stereocampimetric attachment in a specially designed groove in front of the viewing boxes.

Additional refinements are provided, such as ready adjustment of the instrument in see-saw position for testing eyes in upward or downward gaze and raising or lowering the instrument proper.

One of the deficiencies in the ordinary stereoscope is that it is not suitably adapted to excessive degrees of squint. Such a defect may be corrected to a considerable extent by removing all unnecessary barriers in the viewing frame, by providing a more flexible range to the viewing distance and by the use of selective lenses in the viewer. As the unit displacement of the visual axis registers a greater prismatic value for near vision, the stereoscope may be adapted to more marked degrees of convergent squint with either lenses of short focal length (stronger plus lenses) for infinity or lenses of longer focal length for accommodative ranges. If, for example, plus 8 D. spheres are employed in the viewer at infinity range (12.5 cm.), this instrument could be readily adapted to convergent squint amounting to at least 60 prism diopters without the use of supplementary prisms and to divergent deviation of considerably greater amount.

JUNIOR MODEL, OR COMPANION STEREOSCOPE

The companion stereoscope (fig. 2) is, as its name implies, an accompaniment to the senior model. While it was primarily designed to serve as a refined calibrated home-training stereoscope, it is also intended, like the senior instrument, to function as a diagnostic instrument for use in the clinic, although less completely. When both instruments are available, the junior model may be readily adjusted to correspond to the disturbances revealed on the larger instrument, and exercises instituted accordingly. In this sense, it is intended for training purposes.

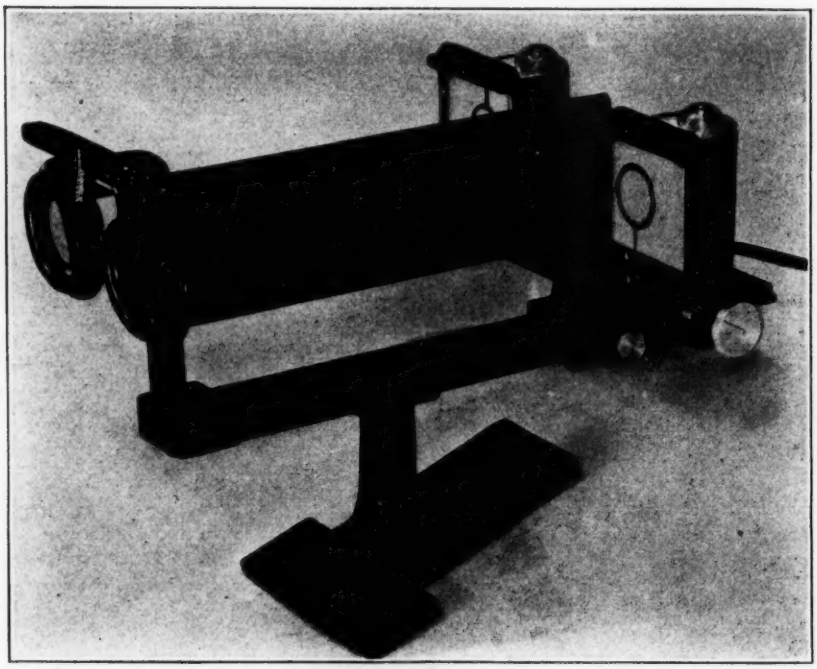

Fig. 2.—The junior, or companion, stereoscope. This photograph is not retouched.

Its features are as follows:

(*a*) Split pictures may be inserted into respective viewing boxes, which may be readily shifted toward or away from each other simultaneously by the turning of a handle that is placed conveniently within reach.

(*b*) The displacement of such pictures may be readily determined by noting the position of an attached pointer which rides with the picture carrier over a finely calibrated millimeter rule that extends from the septum laterally for a distance of 100 mm. While with the senior instrument the displacement of an adjustable slide rule that moves with the

picture carrier is noted in relation to the position of an adjustable stationary pointer and its setting over the fixed rule in order to obtain direct readings, with the junior model readings are made by noting the position of an adjustable pointer that rides with the picture carrier in relation to a single rule (fixed) and deductions are made accordingly. If, for example, the pointer (and picture) moves from an initial setting of 40 mm. to 50 mm., 10 mm. of abduction is recorded; if it moves from 40 mm. to 30 mm., 10 mm. of adduction is recorded.

This pointer, moreover, is adjustable to permit selective settings to correspond to smaller or decentered pictures (as in fig. 2) which travel over greater ranges of adduction or abduction.

(*c*) The viewing boxes, or picture carriers, are individual compartments, each holding a 25 watt candelabra-base daylight bulb and shielded in front by a ground glass for diffusing the light and rendering the illumination of the transparencies quite pleasing. Because of the free opening of each lighting compartment from above, which allows for the exit of heat, the pictures are not heated to any marked extent; nor does the light from the unexposed lamps interfere with the viewing of the pictures. Each picture carrier has its respective flasher.

(*d*) While ordinary paper prints may be used in the machine, it is really intended for transparencies, which prove far more fascinating to the restless child. The advantages of transparencies may be summarized as follows:

First, transparencies allow for a greater range of luminosity, depending on the viewing distance selected as well as on the density of the transparency. The companion stereoscope permits a range of intensity from 0 to almost 100 foot candles—quite an advantage in cases of amblyopia. While this model, unlike the senior stereoscope, does not have a rheostat, a reduction of intensity may be readily effected by inserting a sheet of noninflammable frosted plastocele behind each picture.

Second, transparencies look more natural and lifelike.

For lighting opaque prints or pictures, illumination may be obtained by an accessory table lamp.

(*e*) The size of the picture holder, 6 by 6 cm., was selected to correspond to the most popular sized stereoscopic camera in use at the present time and to answer the requirements of the physician who may choose to make his own pictures.

(*f*) The septum is a unique departure in that it is rigid and immovable and extends the full range of the instrument, so as to insure independent use of each eye at any viewing distance.

(*g*) The connecting rod is clearly marked off in centimeters from 10 to 25 cm., respectively, although the viewing boxes may be brought still nearer, namely, to within 6 cm. of the lenses. This has been made possible through the incorporation of a simple space-saving viewing head which increases the range of accommodation possible.

(*h*) The viewing head consists of two rigid lens holders at an interlenticular separation of 80 mm., which permit the insertion of selective lenses. These lenses are of large diameter, so as to yield a wide angle effect, and are also adaptable to small interpupillary diameters. The frame has a pair of slots or compartments for receiving any additional lenses that may be desired. For example, a person with presbyopia who requires convergence exercises because his reading correction induces an annoying exophoria for near vision (convergence insufficiency) can hardly find inspection through the bifocal reading segments practical. A reading correction added to the viewing lenses (in the stereoscope) solves this difficulty. Prism holders incorporated in the viewing head allow for still greater ranges of elasticity.

(*i*) The companion stereoscope is unusually elastic in its range of operation. By reducing the strength of the viewing lenses, the instrument may be adapted to increasing accommodations. For example, with plus 2 D. spheres, more than 15 diopters of accommodation may be required at the proximal viewing distance of 5.5 cm. In the same way, the possible ranges of adduction and abduction allow for elasticity and control, depending on the viewing distance and the lenses selected in the viewer. In terms of diopters, a total range of 100 diopters or almost 50 degrees, would be possible at a viewing distance of 20 cm., and double this amplitude (in diopters) would be possible at a distance of 10 cm. By bringing the picture to the proximal limits (5.5 cm.), the amplitude may be further increased, not taking into consideration the added reserve amplitude which the prism carriers could provide.

As this instrument, much like its senior partner, is designed to measure not only the range of fusion but the exact amount of its individual components, abduction and adduction, the adjustable pointer operating with the calibrated millimeter scale offers a wide latitude for operation. For anomalies amenable to orthoptic training, such an elastic range is quite generous.

(*j*) The instrument lends itself to the drawing and copying of illustrations by the substitution of ordinary drawing paper in one of the carriers for one of the pictures. This is made possible by the height and angle at which the instrument is built.

The instruments described here were made by the Precise Instrument Company, of Brooklyn.

OPHTHALMOLOGIC SHORTHAND

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The purpose of this paper is to stimulate interest in a standardized ophthalmologic shorthand for use in clinics.

Abbreviations are used by every clinician. Much could be said about the more common ones in use and also about local peculiarities and pet abbreviations. Difficulty in deciphering abbreviations causes a waste of time and energy. The difficulty becomes more pronounced when there is the complication of poor script. The results of this are frequently seen in the permanent records.

Two factors stand out clearly:

1. An accepted ophthalmologic shorthand would make clinical work easier by reducing the time and strain involved in reviewing reports and in recording histories and findings. It should also increase clinical efficiency in the more important work of examining and treating patients.

2. Stenographic efficiency would be greatly improved. Less time would be consumed in making permanent records.

An incomplete list of abbreviations is submitted for study. Many of these are already in common use. Obviously this list will require improvement.

For the development of this shorthand the following rule should be generally adopted: Single letters should be used whenever logical, but when two words beginning with the same letter are to be abbreviated, frequency and importance of use should determine which word will be abbreviated with a single letter and which will receive one or more additional letters, as

Vision	V.
Vitreous	Vit.

The names of certain structures of the eye, because of importance and frequency of use, should be treated as proper nouns and capitalized.

Cornea ..... C.	Disk ..... D.	Choroid ..... Ch.
Sclera ..... S.	Retina ..... R.	Pupil ..... P.
Lens ..... L.	Iris ..... I.	Fundus ..... F.
Conjunctiva ..... Co.	Vitreous ..... Vit.	

Whenever a compound word or a group of two or three words is used, the two or three letters could be united or a hyphen used to signify such an intention.

Corneoscleral	CS or C-S	Tear Sac	T-S
Left Eye	L-E	Anterior Chamber	A-C
Right Eye	R-E	External Ocular Muscles	E-O-M
Both Eyes	B-E		

Designations of important clinical findings that are frequently mentioned, names of special drugs and certain common terms should be capitalized.

Tactile Tension	T-T	Vision	V.
Red Reflex	R-R	Light	Lt.
Near Vision	N-V	Consensual	Cons.
Distant Vision	D-V	Accommodation	Acc.
		Convergence	Conv.
Homatropine	Hom.	Divergence	Div.
Atropine	Atr.	Lt.-Cons.-Acc. (united)	L-C-A
Main Complaint	M-C		

Many words might be treated as common nouns and not capitalized. In this list are the names of indefinite structures, such as "artery," "muscle" and "membrane"; the names of relatively unimportant structures, such as "puncta" and "cilia"; descriptive words or adjectives denoting direction, such as "superior," "upper" and "nasally"; other qualifying words; the terms "opacity" and "injection," and words regularly used in taking a history. Here, too, the rule for importance and frequency of use should determine the use of the single letter for the abbreviation.

For words in less frequent use, some logical abbreviation should be adopted that is easily remembered. Words used in the diagnosis should be capitalized and usually not abbreviated. Words treated as common nouns include the following:

artery	a.	canaliculi	can.	opacity	opac.
vein	v.	capsule	cap.	moderate	mod.
vessel	vl.	upper	up.	severe	sev.
nerve	n.	lower	lo.	lateral	lat.
muscle	m.	superior	sup.	nasal	nas.
cilia	c.	medial	med.	temporal	temp.
lid	l.	headache	h.a.	injection	inj.
puncta	p.	redness	rdn.		
membrane	mb.	tearing	trg.		

Certain suffixes, such as "-itis," in the interest of lucidity probably should not be abbreviated but merely added to the original abbreviation.

Ch ..... itis
R ..... itis

On the other hand, other suffixes could logically be abbreviated.

Scopy ..... py
Retinoscopy .... R-py

To indicate the plural of words, a hyphen and merely "s" could be added.

arteries ..... a-s

Certain nomenclature will necessarily require interpretation in order to give a clear meaning to its abbreviation. Many ophthalmologists favor the use of "d.d." in place of "p.d." in giving the measurement of fundic lesions. This is sound logic, because the optic disk is usually easily defined while the papilla is relatively ill defined.

Disk diameter.... dd.
Vein width..... vw.

In using the latter abbreviation, one could specify that this should be judged from the vein sector, which extends the short distance from the margin of the disk to the border of the papilla. When the vein is pathologically dilated or contracted, the measurement "vw." would signify the examiner's estimation of the caliber of the vein when normal.

In considering the adoption of an ophthalmologic shorthand, one easily recognizes that a habit once established is broken with difficulty. Certainly, this will apply to many ophthalmologists who have been in practice for a long time.

It is to these leaders that one must look for acceptance or rejection of any change in the system of clinical records; at the same time, one feels confident that if such a change is merited, it will be made.

To be of any practical value, a clinical shorthand must have the stamp of official approval, in which case what must at first seem strange becomes an easily familiar and almost common language. The motivation for its adoption would be to simplify clinical reports, to reduce the drudgery of clinical routine and in general to establish an added factor for increased clinical efficiency.

PROGNOSIS OF BACILLUS WELCHII PANOPHTHALMITIS

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Among the rare if not the rarest of deep infections of the eyeball is that caused by Bacillus welchii. This organism is also responsible for highly virulent and extremely grave infections in other parts of the body, which result in a fatal issue in a high percentage of cases. During the World War the mortality from such infections was extremely high, and even now in civil life the infection causes the surgeon much concern, even if the diagnosis is made early and intelligent care is administered.

There have been no deaths reported to date in cases of infection of the eye with B. welchii, even though enucleation has been done instead of evisceration. In such cases the clinical course is not severe, and complete recovery with negative cultures can be expected in two weeks after removal of the eye or its contents.

Twelve cases have been reported in the literature to date, five in Great Britain, four in France, one in Austria and two in the United States. James [1] made a clinical diagnosis of the infection on the basis of gas bubbles in the anterior chamber and eviscerated the globe. Heath [2] reported a case of panophthalmitis in which positive cultures were obtained and evisceration was performed. Ridley [3] reported the development of gas gangrene and panophthalmitis within twenty-four hours after a penetrating wound of the eye. He eviscerated the eye. Hamilton [4] reported two cases of B. welchii infection of the globe, with evisceration in each case. Chaillous [5] reported two cases, and Darier,[6] two. Enucleation was done in all four cases. Rieger [7] reported a case characterized by cachexia in which the injury was due to a piece of steel in the eye. Evisceration and injections of milk brought about speedy

1. James, R. R.: Ophth. Rev. **29**:161, 1910.
2. Heath, W. E.: Brit. J. Ophth. **13**:576, 1929.
3. Ridley, F.: Tr. Ophth. Soc. U. Kingdom **49**:452, 1929.
4. Hamilton, J. B.: Brit. J. Ophth. **14**:452, 1930.
5. Chaillous: Clin. opht. **7**:265, 1904-1905.
6. Darier, A.: Clin. opht. **12**:227, 1906.
7. Rieger, H.: Klin. Monatsbl.. f. Augenh. **96**:548, 1936.

recovery. Berry,[8] of Brooklyn, had a patient who was hit in the right eye with a chip from a nut, and eighteen hours afterward active panophthalmitis developed. A roentgenogram showed a foreign body 2 by 4 mm. in size and located 20 mm. behind the anterior surface of the cornea, 4 mm. below the horizontal plane and 6 mm. nasally to the vertical plane. A gas bubble filled the whole anterior chamber. After the entrance of a knife into the scleral wound, blood-tinged fluid escaped around the shank of the knife. The next day the eye was enucleated, and cultures were positive for B. welchii.

In 1934 I [9] reported a case of B. welchii panophthalmitis. An automobile mechanic was hit in the left eye while pounding on an axle shaft. He did not seek treatment for about twelve hours, and when seen by me he had an intensely inflamed eye, with beginning hypopyon. There was a small wound in the limbus, and what appeared to be a small bubble was seen in the anterior chamber. A roentgenogram showed a piece of steel 1 by 3 mm. in size and located 10 mm. back in the eye. An incision was made, which filled with a coffee-ground discharge. Severe panophthalmitis followed removal of the piece of steel, necessitating evisceration in twenty-two hours. Cultures were positive for B. welchii. After injections of B. welchii serum plus free drainage, the patient was able to leave the hospital in ten days, at which time cultures were negative.

On Sept. 29, 1937, a Negro came to my office stating that the same morning he was hit in the right eye by a stone while using a pick ax. He was sent at once to St. Luke's Hospital. Examination on the operating table disclosed a large perforating wound of the cornea, a traumatic cataract and some prolapse of the iris. A roentgenogram showed a stone in the vitreous; this was removed and a complete flap operation was done after the prolapsed iris was amputated. When the eye was dressed, after twenty-four hours, it was noted that some dark thin pus exuded at the junction of the flap and also what appeared to be fine bubbles arose on slight pressure. A laboratory report on the specimen from the operating room was positive for B. welchii plus Streptococcus viridans. The contents of the globe were eviscerated at once, and it was noted that complete destruction of the vitreous had taken place in forty hours. After receiving massive doses of B. welchii mixed serum, the patient left the hospital in ten days.

In both of my cases panophthalmitis developed in two days, and destruction of the vitreous was complete in that time. Moreover, in both coffee-ground pus was present and the diagnosis was made partly on the basis of the formation of gas bubbles.

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8. Berry, E. M.: Am. J. Ophth. **15**:1022, 1932.

9. Walker, S.: Bacillus Welchii Panophthalmitis, J. A. M. A. **102**:1561 (May 12) 1934.

In Jordan's [10] textbook on bacteriology it is stated that gas gangrene is nearly always due to mixed aerobes and anaerobes of several species. Cultures of B. welchii do not blacken brain and meat mediums normally, but the presence of metallic iron produces marked discoloration. This fact probably explains the coffee ground secretion which was noted by Dr. Berry in his case and also by me in both my cases.

I am impressed with the rarity of B. welchii in perforating and lacerated wounds of the eye. During the last fifteen years I have operated in over 500 cases of intra-ocular foreign body (steel) and in as many cases of penetrating or lacerated wound of the eye. From this large number of cases the present one is my second case of B. welchii panophthalmitis. Most of my patients are from industrial concerns and are therefore exposed to different types of infection. Why my colleagues in general industrial practice often have cases of B. welchii infection and I do not is a mystery to me. The mortality from such infections in other parts of the body is high, and yet in thirteen known cases of this infection following ocular injury there has been an uneventful recovery in every case. With the venae vorticosae and the deep ciliary vessels in close proximity, and exposure of the meninges through the optic nerve sheath possible, it is difficult to explain why there are no fatal cases. Possibly earlier diagnosis and removal of offending tissue may offer some explanation; yet these factors in themselves are not enough.

CONCLUSIONS

1. Intra-ocular foreign bodies of different types are potential carriers of B. welchii.

2. Early diagnosis is apparently of prime importance. The coffee-ground discharge from the wound and the formation of gas bubbles are nearly conclusive evidence, even without laboratory findings.

3. Panophthalmitis due to B. welchii apparently does not have the grave prognosis that B. welchii infection has in other parts of the body.

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10. Jordan, E. O.: A Textbook of General Bacteriology, ed. 11, Philadelphia, W. B. Saunders Company, 1935.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

THE CONCEPT OF ABNORMAL RETINAL CORRESPONDENCE

A THEORETICAL ANALYSIS

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During the past three or four years the concept of abnormal retinal correspondence or abnormal projection has claimed more and more attention among workers in the field of orthoptic training, with the consequence that testing for abnomal retinal correspondence has become one of the most important procedures in the routine examination of patients with squint. Accordingly, if one is a squinter one of three outcomes seems possible: One may practice complete suppression of one eye; one may use either eye alternatingly, or abnormal retinal correspondence or abnormal projection may develop. Should one happen to belong to the group in which the last outcome has developed, a slow process of reeducation, which, according to some authorities, seems almost impossible, is necessary before it may be hoped to straighten the eyes by any orthoptic measure or even to keep them straight after an operative procedure. If this is so, what condition is found in a patient who has abnormal retinal correspondence or abnormal projection?

DEFINITION

A summary of some typical descriptions of abnormal retinal correspondence or abnormal projection found in the literature is as follows: If a patient has abnormal retinal correspondence or abnormal projection he uses the macula of one eye with a nonmacular area of the other eye; the sensation, however, is projected otherwise than as actually received by either eye. For example, a child with an angle of squint of 20 degrees may fuse two pictures in the synoptophore around zero or much nearer to zero than would be expected from his angle of squint. Because of this phenomenon and because the two maculae are apparently not used together, the child is reported as having abnormal retinal correspondence and abnormal projection. To be sure, the literature offers variations of this theory, and different authors have stressed various aspects of it,

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but all seem to be equally ambiguous in their use of the terminology and in the description of the actual findings. For example, Pugh [1] spoke mostly in terms of false projection and stated the belief that its mechanism is mainly cortical. However, she proceeded to attempt to find a retinal correlate in a "specialized hypersensitive zone lying between the macula and the false projection spot." According to this theory, normal projection can be regained only through a process of desensitizing such areas.

Mayou [2] called abnormal retinal correspondence and abnormal projection the objective and the subjective part of the same phenomenon. Yet in the discussion of the diagnosis and treatment of abnormal retinal correspondence she built her theory and method of treatment on purely anatomic grounds. She maintained that the angle of squint as measured objectively must coincide with the angle at which the patient fuses on the synoptophore. No cure may be expected until this condition is reached, and this may be accomplished by alternate stimulation of both maculae at the projection angle.

According to Travers,[3] from 50 to 60 per cent of persons with convergent squint have abnormal retinal correspondence, and if his interpretation of his findings are correct many may be doomed to remain in that condition all their lives. His view is based on a theory of exact retinal point to point relationship, according to which the smallest deviation of the two retinas from such a relationship would cause serious abnormalities. Whether or not normal retinal correspondence may be regained once abnormal retinal correspondence has developed seems to depend on various factors, the two most important being the age or duration of the defect and the size of the deviation. Travers stated the belief that normal retinal correspondence can be obtained more easily in cases of a high degree of concomitant strabismus than in cases in which there is only a small degree of squint, in which instances it seems almost impossible to repair the damage done. He even said that in cases of squint with an angle of 10 degrees and lower it is impossible to reestablish normal retinal correspondence. His is the most extreme view and apparently embodies a generalization of all the other views. He makes the problem an anatomicoretinal one, and on reading his work one may almost become convinced that abnormal retinal correspondence lies at the bottom of all failure of orthoptic as well as surgical treatment.

1. Pugh, M. A.: A Classification of Cases of Concomitant Strabismus, Based on Etiological Factors, Brit. J. Ophth. **18**:446-451, 1934.

2. Mayou, S.: Principles of Orthoptic Training, Brit. J. Ophth. **20**:360-374, 1936.

3. Travers, T. B.: Comparison Between Visual Results Obtained by Various Methods Employed for the Treatment of Concomitant Squint, London, George Pulman & Sons, Ltd., 1936.

None of the aforementioned writers, who represent the more recent advocates of the concept, have given any clear explanation of the anatomic function of abnormal retinal correspondence. They all seem to assume that because they found that in certain squinters the two retinas do not function in accordance with the established theories of the correspondence of retinal points their findings must be due to an abnormality in retinal correspondence. The theory of abnormal retinal correspondence has been developed from the basic hypothesis that exact retinal correspondence is an objectively demonstrable and invariable fact, and, of course, it will stand or fall in accordance with the degree of the validity of this basic hypothesis.

To a careful reader it should appear by now that this theory embodies some grave inconsistencies. Why are two terms, abnormal retinal correspondence and abnormal projection, used? Are these merely two different names for one thing, or are they two concepts for two distinct phenomena? If so, why are the terms employed interchangeably? What is the meaning of these concepts? What assumptions do they imply, and do the facts agree with these assumptions? Is one justified in assuming that because the two retinas do usually manifest some degree of correspondence any apparent deviation from this rule must be due to abnormality? Can normal retinal correspondence and abnormal correspondence be demonstrated objectively? Is the theory of abnormal retinal correspondence and abnormal projection based on objectively demonstrable facts or on subjective inferences? These are some of the questions which may arise, and it is the purpose of this paper to clear up, if possible, a few of them.

ANALYSIS OF THE CONCEPT IN THE LIGHT OF EXISTING NEUROLOGIC FINDINGS

I shall first analyze the concept of abnormal retinal correspondence to see whether the underlying facts on which this theory has been developed are in harmony with the clinical findings. What are the postulates on which this theory rests? The concept of abnormal retinal correspondence depends primarily on making the fovea a point of reference in relation to which the two retinal images must lie on corresponding points. This means that if the two eyes are apparently in such a position that the images fall on the two maculae so that there is a point to point relationship between the cones of the two foveae and the macular areas the two images will be fused into one composite image, because for every nerve ending on one retina there is a corresponding nerve ending on the other retina, which carries its impulses to the same brain cell. These cells are supposed to be distributed on and from the fovea in symmetrical relationship, so that a given nerve ending at a given point on the fovea or a given point on a nerve fiber

from the fovea has its corresponding nerve cell on exactly the same point in the other retina. But a certain degree of inaccuracy had long been observed, and therefore the theory had been changed to an area to area correspondence, the two areas overlapping in some yet unknown manner on their way to the final brain centers. This being the theory, I shall examine whether the necessary facts to substantiate it exist. Is it possible in any way to demonstrate that the nerve fibers from corresponding retinal areas converge either to a final common path or to a common area in the visual brain center or to show whether the nerve endings on the same area of both retinas terminate in one and the same nerve cell in the brain?

Cushing [4] and Horrax and Putnam [5] have presented some clinical evidence from studies of the effects on the visual fields of lesions of the temporal lobe and of a brain tumor in the occipital lobe. This material seems to point to the fact that corresponding retinal points are not represented in the same parts of the optic radiations. If a tumor of the temporal lobe involving parts of the radiation may produce scotomas differing markedly in form in the two eyes, such exact retinal or cortical correspondence as was assumed would certainly not be possible.

Minkowski [6] in a study of monkeys observed degeneration in alternate zones of the lateral geniculate nucleus after enucleation of one eye. His observations indicate that fibers from corresponding points of the two eyes do not reach a final common path up to that zone. He also noted that each retinal point is projected to a relatively large cortical area and that the cortical fields for adjacent retinal points overlap. According to these observations, anatomic evidence for fineness of difference in retinal projection seems to be lacking. There appears to be a good bit of overlapping of separate retinal areas.

However, Lashley [7] recently published two studies on the projection of the retina on the primary optic centers, which furnish considerable evidence in favor of retinal correspondence and correspondence of cortical areas but at the same time allow or account for the apparent absence of cortical correspondence noted by previous investigators. In

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4. Cushing, H.: The Field Defects Produced by Temporal Lobe Lesions, Brain **44**:341-396, 1921.

5. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumor, Brain **55**:499-523, 1932.

6. Minkowski, M.: Ueber den Verlauf, die Endigung und die zentrale Representation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201-252, 1920; **7**:268-303, 1920.

7. Lashley, K. S.: (*a*) The Mechanism of Vision: VII. The Projection of the Retina upon the Primary Optic Centers of the Rat, J. Comp. Neurol. **59**:341-373, 1934; (*b*) The Mechanism of Vision: III. The Projection of the Retina upon the Cerebral Cortex of the Rat, ibid. **60**:570-576, 1934.

these studies small cortical lesions were followed up to the geniculate body within the geniculate nucleus. In the first of these studies [7a] Lashley reported his observation that the optic fibers have no orderly arrangement in the chiasm and optic tract. They intermingle and cross each other freely but finally resume their original relative position, although now inverted and projected in a third dimension in the lateral geniculate nucleus. Within the optic radiation the projection fibers are again complexly crossed, but again they emerge in the cortex after another inversion to reproduce the topographic arrangement of the retina. In the second of his studies [7b] he has given a fairly detailed picture of the cerebral cortex of the rat, and the results clearly indicate that these areas are limited and do not overlap much. He stated:

> The most anteromesial part of the area striata represents the dorsal median of the eye. Along the lateral margin of the area successive sections of the temporal quadrants are represented, and the projection of the nasal quadrants continues along the mesial border. Consecutive zones in the retina seem to be represented by consecutive zones in the area striata. The binocular field is projected to the anterolateral margin of the area.

From these observations Lashley concluded that in the rat there seems to be true cortical representation of retinal areas, with maintenance of a general topographic relation, but that in addition there are considerable plasticity, equipotentiality and mass action. This double observation may explain the apparently contradictory results obtained by different investigators. There seem to be two distinct types of organization which influence and determine visual integration, the one being plastic, equipotential and able to function in a fairly general way and the other having in the cortex a pattern of excitation corresponding in its spatial projection to the spatial pattern of excitation on the retina. These two types of integration have a certain dynamic relationship and seem to be equally important for the integration of the visual experience.

The problem of obtaining neurologic information on the correspondence of the two ocular images is therefore not simple. Perhaps that is why the nature of retinal correspondence is so little understood and has remained on a purely theoretical basis since the time of its formulation by such eminent physiologists as Mueller, Hering and Helmholtz. The importance of retinal correspondence and disparity in relation to fusion and as criteria of depth has long been recognized. Although retinal correspondence seems to be important for fusion, retinal disparity seems to be equally important for depth perception. These two factors have to work hand in hand in order that a person may obtain maximal efficiency in the perception of space.

Many investigators attempted to gain insight into the nature of retinal correspondence through a study of depth perception. Depth, it is assumed, is determined by small degrees of retinal disparity, and the

disparity may be measured in terms of the degree of failure of identical points to be imaged on corresponding points. If the disparity remains within a certain limit, depth is experienced. If it exceeds that limit, diplopia results. This, however, is seldom noticed in ordinary life unless the disparity cannot be overcome by a good "fusion impulse." The most widely accepted theory of depth is thus built on the assumption that a certain amount of disparity exists, and it stresses the importance of disparity rather than that of correspondence.

Another method by which retinal correspondence is frequently studied is with the horopter, the theory of which is undoubtedly known to any student of physiologic optics and therefore needs no mention other than calling to mind that the work of Müller, Helmholtz and Hastings belongs among the classic investigations made with the horopter. In addition to these classic studies, some recent investigations with the horopter have been reported which merit specific mention in this connection, because the investigators, Ames, Ogle and Gliddon [8] stated the belief that their concept of the horopter differs markedly from the classic one and that their horopter furnishes a more adequate and scientific means for studying retinal correspondence. These authors determined the horopter on the hypothesis that "any particular pair of corresponding retinal points by innervating a single brain center gives the subjective sensation of a particular directional value. Moreover, this directional value is the same whether that brain center is innervated by both of the corresponding points or by either alone. Therefore it follows that a relation must exist between the optical projections of retinal elements of each eye and directional values and that these two relations of each eye must correlate through the horopter." Thus, if the projection point and the subjectively projected horopter coincide, the images of the fixation point fall on corresponding retinal cells; but if the true longitudinal point is displaced, this indicates that the images of the point of fixation fall on noncorresponding retinal points.

This definition of the horopter has one advantage over the older ones. It admits that determination of the horopter is based on subjective inferential data without assuming that these data express an exact anatomic retinal correlate. These authors correctly recognized that exact anatomic correspondence cannot be obtained. However, they replaced that assumption with a hypothesis which presents additional difficulties. According to their view, a subject's localization represents a definite response to a specific cortical direction value, this direction value being a sensation due to cortical innervation of a pair of corresponding retinal points. This hypothesis has no more possibility for objective verification than has the other one.

8. Ames, A.; Ogle, K. N., and Gliddon, G. H.: The Horopter and Corresponding Retinal Points, J. Optic Soc. America **22**:575-631, 1932.

In order that the complexity of the problem of determining the nature of retinal correspondence may be fully understood I shall recapitulate the hypothesis which by logical necessity must underlie such a study and discuss the probability of an experimental verification of this hypothesis. The theory of the horopter demands two major assumptions: The first is that if the two retinas are brought into superposition the two areas will have pairs of points which coincide. The second is that if the lines of regard pass through the retinal centers they will meet at the objective point which is fixated; that is, if the projected, or subjective, visual object coincides with the fixated object its images are seen as one, and the retinal points coincide, but if the objective and the subjective visual object do not coincide, diplopia is experienced.

The first of these assumptions can probably never be tested. Before one could know the exact correspondence between the two retinas and the cortical areas one would have to know the exact points of intersection of the projection paths and the exact position of the two retinas. The position of the two retinas could hardly be tested, since, according to the findings of Park,[9] it does not remain stationary. The two eyes have an ever varying angle between the visual axis and the optic axis, and in addition they retract and protrude in their sockets, even during apparent complete fixation.[10] Furthermore, there exist also varying intrinsic movements of the extra-ocular muscles, even though the eyes are steadily fixating on a given spot. Such findings would necessitate an explanation of retinal correspondence based on some dynamic concept rather than depending on a static cell to cell relationship. Although the exact anatomic correspondence of retinal points has no direct observable basis in fact, the subjective, or projected, visual object has repeatedly been used as the criterion of measurement of retinal correspondence by assuming that if a given subject reports any two projected visual objects from the two eyes as coinciding at the fixation point the two retinal points correspond. If, on the other hand, diplopia is reported, it has been assumed that the two images fall on noncorresponding points. That a subject's report cannot be thus relied on Lau [11] pointed out in a paper dealing with binocular depth localization. He found a disparity between the position of the fovea and the projected image and said: "It appears from this that in normal life we probably always

9. Park, G. E.: An Investigation of the Angular Relation of the Visual (Visierlinie) and Optic (Corneal) Axes of the Eye, Am. J. Ophth. **19**:967-974, 1936.

10. Park, R. S., and Park. G. E.: The Center of Ocular Rotation in the Horizontal Plane, Am. J. Physiol. **104**:545-552, 1933.

11. Lau, E.: Binocular Depth Localization, Ztschr. f. Sinnesphysiol. **53**:1-35, 1931.

assume an eye position which deviates from the mathematical convergence." This raises the problem of projection, which will be taken up more fully at a later point in this report.

So far it has been found that the clinical and neurologic evidence furnishes little definite proof as to the exact nature of retinal correspondence. It may merely be stated that some neurologic findings point toward a certain correspondence between retinal and cortical areas but that at the same time considerable equipotentiality and mass action exist. At present no reliable objective method for measuring retinal correspondence is known, and the subject's report of his projected visual experience fails to be very reliable as a criterion of measurement. It is doubtful whether a subject's report of his projected visual object represents an exact replica of his retinal and cortical pattern of the visual object. Since it is almost impossible to obtain an objective measure of normal retinal correspondence, it would be justifiable to assume that measuring abnormal retinal correspondence would be still more difficult. At any rate, any existing disparity between the two retinas of a pair of squinting eyes should be considered in the light of all the knowledge about the nature of retinal correspondence, and such a procedure would probably disclose that the correspondence of the two retinas, whether in normal eyes or in squinting eyes, is exceedingly fluctuating but at the same time follows similar tendencies.

SOME EXPERIMENTAL EVIDENCE

A comparative study which is at present being carried on at the department of ophthalmology of the Northwestern University Medical School gives some evidence indicating that no person, whether he has normal eyes, phoria or squint, fuses on the synoptophore at exactly zero of arc or at exactly the degree of his phoria or the angle of squint.

So far, I have carried out studies on three groups of patients consisting of forty persons without muscular imbalance, forty with either esophoria or exophoria and forty with either esotropia or exotropia. None of these subjects had any pathologic changes of the fundi, and in cases of refractive error all the tests were made with the subject's correction. All these subjects were tested for phoria by means of the screen test and the parallax test, and in cases of strabismus the manifest deviation was measured with the perimeter. For each person four or five measures of retinal correspondence or noncorrespondence were made, with either the amblyoscope or the synoptophore. The subject was allowed to make his own adjustments of the synoptophore for superimposing the two pictures. The results of these subjective tests were then compared with those of the screen test and the parallax test or with those of the perimeter test.

Theoretically, exact correspondence would exist when the measures of the retinal correspondence and the size of the phoria or the degree of squint exactly coincide. According to this definition, any deviation from these measures would indicate abnormal retinal correspondence. The variability or deviation between the degrees at which a person superimposes on the synoptophore or amblyoscope and his measure of the phoria or the degree of squint may be considered a criterion of retinal correspondence and the size of this deviation a measure of retinal noncorrespondence. Each subject received four or five trials with the synoptophore or amblyoscope, in that way giving several measures of retinal correspondence or deviation from retinal correspondence. The largest and the smallest deviation were then tabulated. The average of all these deviations for the three groups of subjects are reported in tables 1 and 2. Thus the results shown are averages for the group.

TABLE 1.—*Average of Horizontal Deviation from Zero in Subjects with Normal Eyes and in Those with Phorias and from the Degree of Squint in Subjects with Strabismus*

Type of Subject	Largest Average Deviation, Degrees	Probable Error of Largest Average Deviation	Smallest Average Deviation, Degrees	Probable Error of Smallest Average Deviation
Normal	3.00	±0.21	1.21	±0.15
With phoria	6.05	±0.41	3.70	±0.34
With squint	6.85	±0.59	4.21	±0.55

Table 1 gives the average of the largest and the smallest horizontal deviation from zero for persons with normal eyes and persons with phoria and from the degree of squint for persons with strabismus. It may be noticed that none of these groups showed exact retinal correspondence. There is considerable variation in the measures of actual superposition on the synoptophore or amblyoscope and that of the phoria or the squint, or of the zero point in cases of normal eyes. This deviation varies from time to time and from person to person, but, considering the subjects as a group, the variability is about the same for persons with squint and those with phoria, while it is somewhat less for persons with normal eyes.

The degree of variability in retinal correspondence not only seems to vary according to the type of the patient—a patient with normal eyes, one with phoria, or one with squint—but it is influenced also by the method of measurement. For example, when the synoptophore was used to measure retinal correspondence in cases of squint, the largest and smallest average deviations were greater than the measures obtained with the amblyoscope. For the synoptophore the largest average deviation was 7.70 degrees, and the smallest 5.60 degrees, while with the amblyoscope the two measures were 5.00 and 2.85 degrees, respectively.

Again, the averages changed if the objective measures, or those determined with the perimeter, were first corrected for the angle gamma before being compared with the measures made with the synoptophore, or the measures of retinal correspondence. This procedure resulted in still smaller averages for the group.

Still another comparison was made, the results of which are given in table 2. As is seen from table 1, the average variability of retinal correspondence in the cases of phoria is 6.05 degrees and 3.70 degrees for the largest and the smallest deviations, respectively, if the deviations are measured from zero. But if the degree of the phorias is taken as the base, the averages for the group are only 3.30 and 2.35, which are practically the same as those for the group of normal subjects. In table 2 the largest and the smallest average deviation from zero for the group of persons without muscular imbalance are compared with the same average deviations for persons with phoria if these deviations were computed from the angle of the phoria instead of from zero. Thus

TABLE 2.—*Average of Deviation from Zero in Subjects with Normal Eyes and from the Angle of Phoria in Subjects with Phorias*

Type of Subject	Largest Average Deviation, Degrees	Probable Error of Largest Average Deviation	Smallest Average Deviation, Degrees	Probable Error of Smallest Average Deviation
Normal	3.00	±0.21	1.21	±0.15
With phoria	3.30	±0.37	2.35	±0.42

considered, the variability from perfect retinal correspondence becomes the same for each group. This indicates that whether or not the findings show normal or abnormal retinal correspondence may depend largely also on the way of treating the data.

Therefore it may be said, in a general way, on the basis of these findings, that no person, whether he has normal eyes, phoria or squint, fuses on the synoptophore or the amblyoscope at exactly zero of arc, the angle of squint or the degree of his phoria. There is considerable variability from person to person, as well as from time to time for a given person. Furthermore, the degree of the discrepancies, if such are present, varies according to various factors, such as the method of measuring the deviation, the method of measuring retinal correspondence, the method of treating the data and probably still other as yet unknown factors. No evidence of any very large discrepancies has been found so far, except in a few instances in which the patient had a false macula. This term is understood to describe the condition in cases of monocular squint associated with amblyopia in which the amblyopic eye fixes at the angle of squint even when the normally fixing eye is covered. At present, our data obtained for patients with strabismus, those with

phoria and those with normal eyes indicate that the abnormality of retinal correspondence found in cases of strabismus is little different from a certain variability also found in persons with normal eyes. This instability is probably due to some action of the movements of the ciliary muscles and extra-ocular muscles the purpose of which is to maintain and establish clear and single vision by focusing the central image on the two foveae.

Although this interpretation of abnormality in retinal correspondence differs considerably from that of other investigators, it is not without experimental support. McAllister,[12] Gertz,[13] and Grim [14] all reported definite movements of each eye when fixation was apparently stationary. Both Grim and Gertz found excursions of from 4 to 10 degrees when the eyes were fixed, while Park [9] reported excursions as high as 17 degrees. They vary from person to person and from condition to condition. They seem to increase with the length of fixation and fatigue and may be altered in nature by administering physostigmine salicylate or atropine sulfate. They may be present as gradual sliding movements or quick and jerky excursions. Park stated the belief that in binocular foveal fixation the physiologic function cannot be steady and constant, since the organism is constantly adapting itself to ever varying neuromuscular-glandular conditions. Only the resultant, the fact that the two foveae are kept steady under the central image, will remain constant.

Evidently the entire idea of retinal correspondence has been taken too literally. It seems that images may well be fused at unequal distances or even at opposite sides of the fovea. Peckham [15] found this to be true in a study in which he made some intricate measurements of the location of the images in relation to the fovea. He noticed that in many cases of normal vision objects are not fused by means of the anatomic corresponding points, that frequently the two foveae are not under the two retinal images. Sometimes there were differences of 4 or 5 degrees, and yet the subject reported single binocular vision. By placing prisms base out or base in before the subject's eyes the author was able to bring the subjects to the point where diplopia should occur, while the latter still reported single vision. Furthermore, the eyes of the subjects in overcoming a 10 degree prism, moved through angles sometimes less than half this amount, while at others they moved even

12. McAllister: The Fixation of Point in the Visual Fields, Psychol. Rev. (supp.) **7**:17, 1905.

13. Gertz, H.: Ueber die Blick-Aberration und ihre Beziehung zur Netzhaut Correspondenz, Acta ophth. **13**:192-223, 1935.

14. Grim, C.: Ueber die Genauigkeit der Wahrnehmung und Ausführung von Augenbewegungen, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **45**:9, 1910.

15. Peckham, R. H.: Foveal Projection During Ductions, Arch. Ophth. **12**: 562-566 (Oct.) 1934.

too far to bring the foveae beneath the image. Bielschowsky [16] in a recent discussion of the etiology of squint reported some observations about retinal correspondence which would indicate considerable fluctuation and dynamic variability. He found that a patient may sometimes show abnormal correspondence while at other times he has normal correspondence, and that this condition is very unstable and may change either spontaneously or in the course of time.

A NEW HYPOTHESIS

If this is so, one may question the right to explain fusion as well as projection in terms of a static anatomic neural function. Whatever the retinal correspondence between the two eyes may be, whether the existing symmetry is geometrically exact, relative and fluctuating, it would be far better to consider fusion and projection as perceptual processes. Fusion would be the cortical interpretation of double images into a single experience, and projection the subjective interpretation of the visual system's constant adaptations to ever varying and complex stimulating conditions. Fusion, according to this definition would have a cortical basis and would be mostly learned. Helmholtz [17] questioned the theory of specificity of nerve function and stated the belief that fusion is a perceptual affair arising from cortical integration at the point to which consciousness is adjunct.

A tiny infant has no projection. His visual objects are not located out in space. Only gradually through manipulating his visual objects do they become associated with his tactual, kinesthetic and auditory experiences and are then given attributes of space. One's knowledge of physical objects is dependent on the composite results of various coordinated sensory experiences. The location of the visual object is inferred from one's localizing movements and auditory and tactual sensations, the experience of parallax, one's knowledge of overlapping objects, the atmospheric conditions and other factors which contribute to knowledge of space. One naturally assumes that all the different sensory objects have the same location. Thus one learns to see things where one hears them and to hear them where one sees them, and one sees them in the place where one's hands can touch them.

As has already been mentioned in an earlier part of the paper, the assumption that the visual object must be located in the same position as its retinal image is without justification. For further clarification, let the case of ordinary visual sensation be considered. Its existence

16. Bielschowsky, A.: The Etiology of Squint, Am. J. Ophth. **20**:478-489, 1937.

17. Helmholtz, H.: Treatise on Physiological Optics, translated from German edition and edited by James P. Southall, Rochester, N. Y., The Optical Society of America, 1925, vol. 2.

and nature are functions of the external object that reflects the light into the eye. Its quality is dependent on the intensity of the light and the nature of the light waves. It is also dependent on cortical and retinal activities and the refraction by the lens through which the light is transmitted plus certain changes of the position of the eyes in their sockets due to retracting and advancing movements. Thus the visual object is dependent on a long series of events which are bound to modify the exact shape and position of the object. Furthermore, one localizes a visual object as right side up, while it is well known that the refractive surfaces of the eye give an inverted retinal image of all external objects. It has also been shown by Stratton [18] that the wearing of a pair of lenses which rotates the retinal image 180 degrees does not materially affect the visual experience. Although these lenses inverted the visual world in respect to the tactual and conceptual standards, a new system of habits of manipulation was soon established which brought about a readjustment of the visual standards, and after a few days of practice the world once more became right side up. Thus one's interpretation of one's visual experience is not at all fixed by the actual sensory organization. In regard to position and shape, one's visual object is not a projected replica of one's retinal image but a perceptual and interpretative phenomenon.

The process of the retinal correspondence of the two eyes may be explained in a similar fashion. At birth the correspondence between the two retinas is very poorly developed. By watching an infant's eyes little coordination of the two eyes can be observed.

The learning of ocular coordination differs little from that of any other function of coordination, such as walking and swimming. At first the child exercises his muscles by trial and error, and the result of these random movements is clear, single vision. Gradually he learns to repeat these movements, which at first more or less accidentally brought clear and single vision, until gradually he learns to associate a certain muscular pattern with clear and single vision.

This initial clumsiness in a child's ocular coordination is easily understandable if one considers the fact that most children are born somewhat hyperopic, owing to axial development of the eye. At any rate, this condition brings about the necessity for an ever changing adjustment of the neuromuscular integration. Furthermore, one frequently finds that in infants the internal rectus muscles are hypertonic. All these conditions must influence the process of coordination and complicate the adaptive function, so that it is not surprising that the development of coordination is a gradual process which requires considerable learn-

18. Stratton, G.: Upright Vision and the Retinal Image, Psychol. Rev. **4**: 182-187, 341-360 and 463-481, 1897.

ing. Thus, through the process of repeated reciprocal innervation and inhibition of the tonus in the opposing muscles, which is partly an innate and partly a learned function, the organism produces a tendency of the visual fields to unite, and the child learns to fuse the separate retinal fields.

Through association of movements of convergence and the innervation of the ciliary muscles, which are purely proprioceptive processes, the child is building up the "accommodative convergence association." But at this state he still has no fusion. So far his visual behavior is mainly a proprioceptive space direction affair, with poor coordination, resulting either in overconvergence or overinnervation of this ciliary muscles, or vice versa. This consequently causes diplopia and blurred vision, which sooner or later the child recognizes. Whenever this happens the child learns it from his association with touch. He discovers that when his eyes tell him that there are two objects his hands tell him that there is only one. This stage is probably reached about the time when he is getting old enough for the need of more accurate and clearer vision. Now a higher integration comes into play. Through voluntary attention he learns to control indirectly his internal rectus muscles, and also to control indirectly his ciliary muscles, because of their associated function with convergence.

This process may be described in mechanistic or subjective terms. In mechanistic terms the description is as follows: Whenever the two retinas are stimulated by light the equilibrium of the existing photochemiconeuromuscular integration becomes upset. As a consequence a new process of integration comes into play in order to reestablish equilibrium. This integrative behavior is in the nature of a proprioceptive convergence-accommodation adjustment, plus the cortical or associative direction process. In subjective terms, one would say that the subject adjusts his eyes by means of directing attention until clear and single vision is obtained, or until he fuses. This fusion becomes the subject's interpretation or perception of a specific image and response pattern of his visual apparatus.

By means of manipulating an object by the process of coordinated movements of the extra-ocular muscles and by the constant shifting of the adjustment of the intra-ocular and ciliary muscles the subject gathers valuable clues for his interpretation of the location of visual objects. He learns that if his eyes are in a certain position and the image is on a certain part of the retina, the object lies straight ahead, to the right or to the left, up or down.

Should something suddenly happen to upset this neuromuscular balance, the person would at first continue with the same movements for projection or localization, which are the only ones which he knows how to use. But now, since drastically new conditions have arisen, his

interpretative responses no longer bring the habitual results. He experiences diplopia, with the consequence that his visual world disagrees with his tactual and auditory one. Since the human organism is an adjusting one, it immediately starts to develop a new object space pattern based on sensory experience. In other words, the person learns to adjust the new clues to the standards of his other modalities. He knows from his experience of touch or sound where an object should be, and to that knowledge he fits the ocular clues, by suppressing one eye, by using either eye alternatingly or by what some investigators have called false projection.

To call this new localization false projection is unjustifiable, since from the subject's perceptual standard this projection is as correct as his earlier one. It is false only from the observer's point of view, who assumes that the retinal image and projected experience must coincide. That such an assumption is false the results of experiments of Gertz, Peckham and Stratton give ample proof. The projection or interpretation of the visual experience is not bound by the exact sensory phenomena. Perception and judgment are complex behavior patterns of which the sensory elements are only one aspect. Perceptual activity is influenced by past experience and not only by a specific sensation received at a particular moment, and therefore the resulting behavior, whether it be in the form of a verbal statement or in the form of a localizing response, would necessarily embody a broader and more complex neuromuscular pattern than would be the case if it were only a direct response to a simple sensation. Accordingly, one cannot assume that the localizing into space of a visual object is an exact projection of the two retinal images of that object.

In the same way one can explain why sometimes after an operation for squint the patient localizes or fuses two images at the angle of his squint, although his eyes are now straight. This condition of *horror fusionis* usually soon disappears, which is added evidence that the organism quickly learns new localizing responses.

CONCLUSIONS

I shall now return to the problem of abnormal retinal correspondence and projection. As was shown in the course of this paper, it is difficult to study the nature of abnormal retinal correspondence, because so little is definitely known about normal retinal correspondence. The classic assumption that the two retinas correspond exactly by either a cell to cell or a retinal to cortical area relationship has no neurologic proof so far beyond the discovery of a certain topographic arrangement of the retinas in the cortical centers. Furthermore, various results of studies of fusion and projection carried out on subjects with emmetropic eyes

and without muscular imbalance have shown much more disparity between any given images on the two retinas than could possibly be present were there a geometrically exact correspondence of the two retinas. The accumulated body of facts points toward a dynamic plasticity rather than toward a static anatomic relationship.

It has also been questioned whether in investigations of abnormal retinal correspondence in which the physician questions his patient about the nature of the visual projected images one may assume that the patient's report exactly represents the image patterns on his retinas. The fact that fusion and projection are perceptual and judgmental behavior, which are influenced by past experience and experience from other sense modalities, suggests that a patient's verbal or localizing report of projected images cannot be considered a reliable criterion for measuring retinal correspondence or the degree of abnormal retinal correspondence. It must be remembered that a patient's localizing of his images is a perceptual interpretation of his visual experience, his conditioning by past experiences, and his knowledge of the object from touch and audition, as well as from the clues of his ocular convergence-accommodation adjustments.

The concept of retinal correspondence has probably been taken far too literally. Since analysis of the actual facts indicates considerable plasticity of retinal correspondence in normal eyes, it may well be assumed that those discrepancies in retinal correspondence found in certain cases of squint are of a similar nature and should therefore be explainable by the same hypothesis. Such a dynamic and functional approach to abnormal retinal correspondence in cases of concomitant strabismus would probably change the problem from one of abnormality to one of variability of the normal.

Drs. Sanford R. Gifford, G. E. Park and G. Guibor gave opinions, observations and helpful suggestions in this study.

News and Notes

EDITED BY W. L. BENEDICT

SOCIETY NEWS

Scientific Meeting, Eye Section, Philadelphia County Medical Society.—At the meeting of the Eye Section of the Philadelphia County Medical Society held at the Philadelphia County Medical Society Building, Feb. 3, 1938, Dr. Louis R. Wolf reported on clinical cases from the Temple University Hospital. The following papers were read: "Visualizing the Pathogenesis of Senile Cataract," by Dr. Aaron Brov; "Anatomy and Functions of the Orbital Fascia," by Dr. Charles W. LeFever, and "Differentiating the Various Types of Blurred Disks," by Dr. Glen G. Gibson.

At the meeting held on March 3, 1938, at the Philadelphia County Medical Society Building, Dr. Irvin Levy reported on clinical cases from the Will's Hospital. Dr. William Zentmayer discussed "Etiology and Diagnosis of Intraocular Tumors;" Dr. Alexander Fewell, "Prognosis and Treatment of Intra-ocular Tumors," and Dr. Edgar W. Spackman, "Ocular Roentgenologic Diagnosis."

Montana Academy of Ophthalmology and Oto-Laryngology.—The semiannual meeting of the Montana Academy of Ophthalmology and Oto-Laryngology was held in Butte, February 13 and 14. Dr. Roy Grigg, of Bozeman, was elected president for the ensuing year, and Dr. Ashley Morse, of Butte, was elected secretary-treasurer.

Royal College of Physicians and Surgeons of Canada.—At a recent meeting of the Royal College of Physicians and Surgeons of Canada it was decided to establish boards for certification in a number of specialties, including ophthalmology. A committee composed of Dr. S. Hanford McKee, of Montreal, chairman, with Dr. W. H. Lowry, of Toronto, and Dr. Septimus Thompson, of London, Ontario, was appointed to draft recommendations for future consideration.

Dallas Southern Clinical Society.—The tenth annual spring clinical conference of the Dallas Southern Clinical Society was held in Dallas, March 14 to 17, 1938. The headquarters were at the Hotel Adolphus. Lectures in ophthalmology were given by Dr. Conrad Berens, of New York.

Philadelphia County Medical Society.—At the meeting of the Eye Section of the Philadelphia County Medical Society on Feb. 3, 1938, the following program was presented:

"Clinical Cases from Temple University Hospital," by Dr. Louis R. Wolf; commentator, Dr. Walter I. Lillie.

"Visualizing the Pathogenesis of Senile Cataract," by Dr. Aaron Brav; commentator, Dr. Leighton F. Appleman.

"Anatomy and Functions of the Orbital Fascia," by Dr. Charles W. Le Fever.

"Differentiating the Various Types of Blurred Optic Disks," by Dr. Glenn G. Gibson, with a lantern demonstration.

Before the program a postgraduate conference in ophthalmology was held by Drs. L. C. Peter, A. G. Fewell and I. S. Tassman.

New York Eye and Ear Infirmary.—The annual postgraduate week of the School of Ophthalmology and Oto-Laryngology of the New York Eye and Ear Infirmary will be held March 28 to April 2, 1938. Courses will be given in ophthalmology, otology, rhinology, laryngology, bronchoscopy and esophagoscopy. During the course, daily luncheons will be held, at which speakers of the day will preside. The annual dinner will be held Friday evening, April 1, at 7:30, Dr. Clyde E. McDannald presiding. Daily demonstrations will be held in the various clinics and in the special departments of the institution.

The fee for the entire course will be $50. Application for registration should be mailed to the School of Ophthalmology and Oto-Laryngology, New York Eye and Ear Infirmary, Thirteenth Street and Second Avenue, New York.

Chicago Ophthalmological Society.—At the annual meeting of the Chicago Ophthalmological Society the following officers were elected for the year 1938-1939: president, Dr. Georgiana Dvorak-Theobald; vice president, Dr. Leo L. Mayer, and councilor, Dr. E. G. Nadeau. Dr. Earle B. Fowler was reelected secretary-treasurer.

Dr. C. S. O'Brien, of Iowa City, assisted by Dr. James Allen, read a paper entitled "Staphylococcus Conjunctivitis."

Eye Health Committee.—Plans for a study of problems pertaining to the health of the eyes of college students are being made by the Eye Health Committee of the American Student Health Association, with the aid of an Advisory Committee from the American Academy of Ophthalmology. The project will have the cooperation of the National Society for the Prevention of Blindness.

Dr. R. W. Bradshaw, of Oberlin College, Oberlin, Ohio, is chairman of the Eye Health Committee of the American Student Health Association; Dr. W. L. Benedict, of Rochester, Minn., is chairman of the Advisory Committee from the American Academy of Ophthalmology, and Dr. Anette M. Phelan represents the National Society for the Prevention of Blindness.

Dr. Bradshaw's committee includes Dr. Lee H. Ferguson, of Western Reserve University, Cleveland; Dr. L. M. Hickernell, of Syracuse University, Syracuse, N. Y., and Dr. Ruby L. Cunningham, of the University of California, Berkeley.

The inadequacy of present methods of discovering college students who need ophthalmic attention has impelled the American Student Health Association to seek the active help of ophthalmologists in dealing with this problem, according to Dr. Bradshaw.

Serving on Dr. Benedict's committee of ophthalmologists are Dr. LeGrand H. Hardy, of New York, and Dr. Harry S. Gradle, of Chicago.

GENERAL NEWS

Sight Saving Week.—The Sight Saving Council of Memphis, Tenn., in which the ophthalmologists of the city cooperated, sponsored a Sight Saving Week in Memphis, January 10 to 14, during

which pertinent information pertaining to conservation of vision from the medical and the practical standpoints, including those of accident prevention, illumination and industry, was dispersed by lectures and radio to a vast audience. More than ten thousand persons were personally addressed by Mr. Milton Bowman, of Cleveland, who used slides pertaining to the conservation of the eye in relation to traffic and industrial hazards.

Course in Ophthalmology.—An intensive course in ophthalmology will be given by Dr. Sidney Olsho and Dr. L. Waller Deichler, of Philadelphia, April 4 to 9, 1938. Further information can be secured by writing Dr. George W. Mackenzie, 269 South Nineteenth Street, Philadelphia.

American Board of Ophthalmology.—Examinations for certification in ophthalmology will be conducted by the American Board of Ophthalmology in accordance with the following schedule: San Francisco, June 13; Washington, D. C., October 8; Oklahoma City, November 15.

Application for examination should be sent to the office of the American Board of Ophthalmology, 6830 Waterman Avenue, St. Louis.

UNIVERSITY NEWS

Study in Physiologic Chemistry.—The department of ophthalmology of the University of Chicago is making an extended effort to build up an interest in physiologic chemistry in relation to ophthalmology, under the guidance of Dr. A. C. Krause, and will welcome graduate students in this special field.

Obituaries

ISADORE GOLDSTEIN, M.D.
1879-1937

Isadore Goldstein died on Dec. 23, 1937. Turning from the practice of general medicine, he began his career in ophthalmology at the New York Eye and Ear Infirmary in the clinic of Dr. John E. Weeks, who encouraged him in his interest in plastic surgery of the eye. After Dr. Weeks' retirement in 1919, he came under the influence of Dr. John M. Wheeler, who guided his further development in this branch of surgery. He soon emerged as an accomplished surgeon, with a deep interest in and unusual talent for reconstructive work. From 1929, as senior assistant surgeon in Dr. Bernard Samuels' clinic, he was afforded every opportunity to develop and demonstrate his methods of plastic surgery. Extensive procedures requiring several operations were so carefully devised as to require little deviation from the original plan in their execution. Postoperative dressings received the same meticulous care as the operation itself. In a field in which successful results are not the rule, his were unusually numerous. Among the procedures he devised were: recession of the levator muscle for the relief of exophthalmos in cases of exophthalmic goiter, construction of lids for the retention of prosthesis following exenteration of the orbit and the replacement of diseased conjunctiva of the upper lid with buccal mucosa in cases of obstinate vernal catarrh.

Dr. Goldstein's second great interest was ocular pathology, especially in relation to general diseases. In 1928, largely through his efforts, a department of ocular pathology was established at the Mount Sinai Hospital. Numerous articles appeared in the ARCHIVES OF OPHTHALMOLOGY, including those dealing with the histologic picture of melanosis in Recklinghausen's disease, of periarteritis nodosa, of acute lupus erythematosis and of Niemann-Pick's disease and with the effects of irradiation on the eyes of human embryos. In 1932 Dr. Goldstein was appointed ophthalmic surgeon at the Mount Sinai Hospital.

During vacations abroad he was invited to exhibit his surgical skill in Cairo, Paris and Jerusalem. In 1930 he assisted in the establishment of the ophthalmic division of the Hebrew University in Palestine.

He was an indefatigable and conscientious worker, an able teacher, a keen observer who was well informed on many topics in general medicine and a source of comfort to many men in times of "surgical stress."

DAVID WEXLER, M.D.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ARE THE CLUMP CELLS OF THE UVEA DERIVED FROM CHROMATOPHORES? A. BAKKER, Arch. f. Ophth. **137:** 611 (Oct.) 1937.

The clump cells of the uvea may be of ectodermal or of mesodermal origin. The presence of typical clump cells in blue irides, the stroma cells of which contain no pigment, and in fetal irides, in which no uveal pigment has yet been formed, proves the possibility of ectodermal origin. With his method of perfusion of explanted tissue, Bakker has shown that chromatophores under conditions of slightly impaired circulation are transformed into clump cells. Thus, the possibility of a mesodermal origin of the clump cells is also proved.

P. C. KRONFELD.

THE PALPEBRAL FISSURE IN NEGROES. J. L. ROLLIN, Ztschr. f. Augenh. **89:** 95 (April) 1936.

In a study of 800 typical Negroes from many African tribes, Rollin found only 2 whose palpebral fissure sloped down temporally while in eight the outer canthus was definitely higher than the inner canthus. This would disprove the existence of a negroid facial characteristic proposed by Rontils, who states that the outer commissure is typically lower than the inner commissure in the Negro.

H. GIFFORD JR.

Biochemistry

THE SPERMINE BASES OF OCULAR TISSUES. A. C. KRAUSE, Am. J. Ophth. **20:** 508 (May) 1937.

Krause gives the following summary:

"A quantitative analysis of the spermine and spermidine in bovine conjunctiva, sclera, corneal epithelium and stroma, choroid, iris, vitreous humor, retina, optic nerve, and brain was made. There is reason to believe that these substances take a part in some unknown important physiological function of active metabolic tissues."

W. S. REESE.

LACRIMAL ELIMINATION OF DEXTROSE IN HYPERGLYCEMIA INDUCED BY EPINEPHRINE. D. MICHAIL, P. VANCEA and N. ZOLOG, Compt. rend. Soc. de biol. **125:** 1095, 1937.

Hyperglycemia induced by epinephrine is regularly accompanied by the lacrimal elimination of dextrose. The dextrose in the tears is demonstrable in from twenty to seventy minutes after the injection of epinephrine. Lacrimal elimination commences when the sugar content is from 30 to 40 per cent above normal. This fact shows that up to a certain point the threshold of lacrimal elimination of dextrose is lower than that of renal elimination.

J. E. LEBENSOHN.

Congenital Anomalies

COLOBOMA OF THE MACULA LUTEA IN MONOZYGOTIC TWINS: REPORT OF A CASE. K. VOGELSANG, Klin. Monatsbl. f. Augenh. **98:** 322 (March) 1937.

Vogelsang reports in detail the cases of two male twins aged 8 years with colobomas in their right eyes. The boys showed mirror-like asymmetries of their fingers, ears, features, form of palate, teeth and tongue and various other structures. The father's eyes were normal in form and function, except for irregular pupils, deep physiologic excavation of both disks and slight lighter discoloration of the temporal portion of the choroid. Vision was normal in each eye. The mother had juvenile glaucoma, which may constitute a correlation to the malformation in the eyes of the twins. Vogelsang considers the colobomas malformations because of their concordant occurrence in monozygotic twins and because no further changes occurred in the fundi during eight years. The literature in point is cited.

K. L. STOLL.

Cornea and Sclera

SCLEROMALACIA. F. A. KIEHLE, Am. J. Ophth. **20:** 565 (June) 1937.

A 64 year old woman had had rheumatism since the age of 21. She had been bedridden for nine years because of deforming polyarthritis, practically all her joints being affected. When the author first saw the patient, her eyes showed slaty gray scleras, described as "vitrification," the thinned scleras allowing the choroid to show through. The episcleral tissue after a stage of edematous swelling gradually was absorbed. The media were clear and the fundi were normal, but later both lenses became opaque and the eyes sightless, and in recent years multiple radiating perforations occurred in each iris. Kiehle compares this case to others reported in the literature and discusses the relation of the condition to arthritis.

W. S. REESE.

ON MEGALOCORNEA (CLINICAL CONTRIBUTION AND PATHOGENIC CONSIDERATION). V. PATERNOSTRO, Ann. di ottal. e clin. ocul. **64:** 846 (Dec.) 1936.

Paternostro describes three cases of bilateral megalocornea of the familial type, in one of which there was corectopia. He reviews the various theories of the origin of the condition and then attempts to evaluate the embryologic factors. He concludes that megalocornea is produced on the marginal portions of the secondary optic cup by abnormal differentiation.

F. P. GUIDA.

HEMATOLOGIC STUDIES IN CASES OF SYPHILITIC INTERSTITIAL KERATITIS. A. MOTEGI, Arch. f. Ophth. **137:** 527 (Oct.) 1937.

Motegi compares the hematologic findings for patients with syphilitic interstitial keratitis with those of normal controls and for persons with other corneal diseases. He reports an enormous amount of data, which do not lend themselves to abstracting.

P. C. KRONFELD.

CLEARING OF THE OPAQUE TRANSPLANT BY ADDITIONAL KERATOPLASTY. S. WELTER, Vestnik oftal. 10: 643, 1937.

The technic of additional nonpenetrating or penetrating transplantation at the site of the opaque transplant is described. Three eyes in which the transplants were opaque for eleven, thirteen and thirty-five months, respectively, were operated on by Filatov's method of additional transplantation of fresh corneal tissue around the old transplant. All the opaque transplants became semitransparent within two or three months. This procedure gives new hopes as to the possibility of combating the opacification of corneal transplants.

O. SITCHEVSKA.

AUTOHEMOTHERAPY OF CORNEAL ULCERS. A. B. KOLENKO, Vestnik oftal. 10: 820, 1937.

The purpose of this experimental work was to determine the value of autohemotherapy in cases of purulent ulcer of the cornea, its prophylactic action and its mechanism of action according to the morphologic changes of the leukocytes in rabbit's blood. Thirty-six rabbits were used for the experiment. From five to seven injections of from 0.1 to 0.15 cc. of blood per kilogram of body weight were given. The experiment was divided into five parts, as follows: Staphylococcic corneal ulcers were treated with general autohemotherapy (subcutaneous injections) and with local autohemotherapy (subconjunctival injections); corneal ulcers were produced by smallpox vaccine, and the prophylactic action of general and of local autohemotherapy against the staphylococcic infection of the cornea was investigated. Repeated examinations of the blood were done on ten rabbits. An ulcer was produced by the introduction of an emulsion of a pure culture of Staphylococcus aureus into the anterior chamber and into the deep layers of the cornea. The cure of the ulcer was considered complete when it was covered with epithelium. Kolenko came to the following conclusions on the basis of his experiments:

1. A corneal ulcer is best produced in the rabbit by the introduction of an emulsion of staphylococci into the deep layers of the cornea, because the doses can be regulated and excessive irritation is avoided.

2. An experimental ulcer has a tendency to self cure.

3. Smallpox vaccine has no advantage over the staphylococcus emulsion as an infectious material for the production of a corneal ulcer.

4. Subcutaneous or subconjunctival autohemotherapy shortens the duration of purulent keratitis about 8 per cent as compared with the course of a control ulcer.

5. Prophylactic autohemotherapy, general or local, does not prevent the development of an experimental ulcer, but the course is sluggish and the time of epithelization is shortened as compared with these factors in a control ulcer.

6. The number of white and red blood cells and the leukocytic formula in rabbits vary individually. The change of the morphologic picture could not be systematized.

7. Autohemotherapy, local or general, is a favorable factor in the treatment of experimental ulcer.

O. SITCHEVSKA.

Experimental Pathology

THE FUNDUS OCULI OF THE RABBIT WITH VITAMIN A DEFICIENCY. F. SCULLICA and E. FULCHIGNONI, Ann. di ottal. e clin. ocul. **65:** 427 (June) 1937.

Spotted or brown rabbits were placed on a diet deficient in vitamin A. The fundi were observed after dark adaptation of thirty minutes and after exposure of one eye to a light of 500 foot candles for four hours. The fundus of the exposed eye was compared with that of the unexposed eye at various intervals after exposure and with that of a rabbit on a normal diet and treated to the same exposure. In animals on a diet deficient in vitamin A as well as in those on a normal diet, a definite decoloration of the fundus was observed in the eye exposed to light. This decoloration was attributed to bleaching of the visual purple. In animals on a normal diet the color returned to normal in from seventeen to eighteen hours after exposure, while in those on the deficient diet the difference in color persisted much longer, the length of time required for a return to the normal color being proportional to the severity of the deficiency in vitamin A.

In addition to this decoloration, there was seen in normal animals, and more markedly in animals with vitamin deficiency, a change in pigmentation of the fundus after exposure to light. The homogeneous pigmentation of certain areas was replaced by smaller zones of pigment-forming islands and archipelagos of pigment surrounded by clear areas in which the choroidal vessels were clearly seen. The phenomenon was reversible after dark adaptation but became less so in animals with more severe vitamin deficiency. Another phenomenon observed was the appearance of medullated nerve fibers, which extended from the disk to the periphery in all directions after exposure to light. After dark adaptation, this picture was replaced by the normal one, showing the medullated nerve sheaths only near the optic disk. The changes described were all seen at a time before the symptoms of xerosis and keratomalacia occurred.

S. R. GIFFORD.

General

THE EYE HOSPITAL AT SHIKARPUR, INDIA. R. BUXTON, Brit. J. Ophth. **21:** 605 (Nov.) 1937.

Buxton gives a vivid description of the adverse conditions under which operations were performed at the Eye Hospital at Shikarpur at the time Sir Henry Holland took charge in 1910. Under his supervision as medical missionary of the Church Missionary Society at Quetta, conditions nowadays are much better. During a stay of about eight weeks this year of the three visiting ophthalmic surgeons, 3,400 operations, including 1,427 cataract extractions, were performed. On the busiest day the total number of operations was over 170, including 96 cataract extractions. Chronic glaucoma is treated either by Elliot's trephine operation or by a broad iridectomy. On the basis of the results, Holland finds little choice between the two methods. He prefers the intracapsular method for cataract extraction, using a modification of the Smith method.

W. ZENTMAYER.

IS THE ARTIFICIAL EYE A CURATIVE OR AN AUXILIARY REMEDY? C. QUINT, Klin. Monatsbl. f. Augenh. **98**: 347 (March) 1937.

The question whether a glass eye is only an auxilary remedy, desirable for cosmetic effect, or an actual curative agent is of great importance in problems arising in regard to workmen's compensation.

Quint reasons that from the medical standpoint an artificial eye has a predominantly curative value and, secondarily, an auxiliary value. The cure is not finished with the enucleation, because a condition remains which is not physiologic. The lids, being deprived of their support, sink back and exert a constant friction on the conjunctiva. This leads to thickening of the conjunctiva and increased secretion and finally to atrophy of the conjunctiva. Mucopurulent discharge frequently produces eczema of the lids and the adjacent areas, and irritation may be transmitted to the other eye. These conditions can be prevented by constant use of an artificial eye. Therefore, the curative value of a glass eye is equal at least to that of spectacles.

K. L. STOLL.

General Diseases

OCCURRENCE OF SO-CALLED THROMBO-ANGIITIS OBLITERANS IN THE EYE. Y. UYAMA, Arch. f. Ophth. **137**: 438 (Aug.) 1937.

Uyama reports the cases of two patients (a Japanese man aged 24 and a Japanese woman aged 35) who showed pallor of the entire skin, cyanosis of the extremities, hippocratic fingers and toes and circumscribed loss of sensibility on the extremities. Ophthalmoscopically, the picture was that of the late stages of perivasculitis and endovasculitis, arteries as well as veins being involved. Long stretches of retinal vessels were transformed into white strands or accompanied by white sheaths. One eye of one of the two patients became available for pathologic examination, which revealed chiefly proliferation of the intima, new formation of elastic lamellae and no signs of inflammation. These anatomic observations are the same as those described in other organs in cases of Buerger's disease.

P. C. KRONFELD.

Glaucoma

LECTURES ON GLAUCOMA. R. E. WRIGHT, Am. J. Ophth. **20**: 462 (May) 1937.

This article does not lend itself to abstracting and deals with glaucoma particularly as it occurs in India. Wright speculates on the possibility of glaucoma's being systemic in origin and discusses epidemic dropsy, which has primary glaucoma as one of its clinical manifestations. As epidemic dropsy is due to ingestion of a certain type of rice, he infers that glaucoma may be caused by ingested toxins, defective ingestion or abnormal metabolites elaborated in the body from normal foods.

W. S. REESE.

LECTURES ON GLAUCOMA. R. E. WRIGHT, Am. J. Ophth. **20:** 571 (June) 1937.

This lecture on the operative treatment for glaucoma does not lend itself to abstracting. Wright takes up sclerocorneal trephining, the Lagrange decompression, iridencleisis, cyclodialysis and posterior sclerotomy. He discusses the complications and the postoperative treatment of trephining.

W. S. REESE.

PATHOGENESIS AND THERAPY OF HEMORRHAGIC GLAUCOMA, WITH ESPECIAL REFERENCE TO CONTINO'S ANTERIOR FORM. G. FAVALORO, Ann. di ottal. e clin. ocul. **65:** 241 (April) 1937.

Favaloro presents clinical evidence in support of Contino's contention that in certain cases of glaucoma there is a distinct syndrome, which is amenable to mydriatics instead of miotics and in which the eyes are especially prone to hemorrhages. Although the cases reported by Contino are not very convincing, he believes that they aid in separating the usual type of glaucoma, due in his opinion to swelling of the vitreous, from the anterior type. The latter he considers as primarily an angiopathy, the increased tension being secondary to vascular disturbances in the iridocorneal angle. The benefits of scopolamine and atropine in such cases depend on their decongestive effect.

S. R. GIFFORD.

Lens

TECHNIQUE OF CATARACT EXTRACTION DURING NARCOSIS. BASIL GRAVES, Brit. M. J. **2:** 319 (Aug. 14) 1937.

In order to avoid lack of cooperation and mental strain on the part of the patient during an operation for the extraction of cataract, Graves, in addition to the usual local injection of procaine hydrochloride, induces narcosis of a degree sufficient to abolish all consciousness. The state of sleep lasts from seven to fourteen hours after the operation, and the unpleasant after-effects, such as vomiting, abdominal pain, delirium and retention of urine, are thereby eliminated. The author's method consists in the use of paraldehyde rectally, accompanied by a hypodermic injection of a mixture of opium alkaloids (omnopon). Bromides are given by mouth during the preceding twenty-four hours. The pupil is dilated with two swabs of cotton wool soaked, respectively, with a 2 per cent solution of homatropine hydrobromide and a 1:1,000 dilution of epinephrine hydrochloride and inserted under the upper eyelid.

The author also fully describes his modified technic of cataract extraction, which consists of a rather intricate suture of the cornea and the sclera. After the usual section, without iridectomy, a piece of the anterior capsule of the lens is removed with a capsule forceps. The same method of inducing anesthesia is used in cases of glaucoma in which trephination is performed. Illustrations of the author's technic of cataract extraction accompany the article.

ARNOLD KNAPP.

URICACIDEMIA AND CATARACT. G. VILLANI, Ann. di ottal. e clin. ocul. **65:** 306 (April) 1937.

The uric acid content of the blood of sixty patients with cortical cataract was determined. Patients with nuclear cataract were not included in the study. Only two patients were known to be subject to gout. The normal figure was considered to be from 0.04 to 0.05 per cent. Only one patient had a low uric acid content—0.031 per cent. The values for twenty-five patients ranged between 0.051 and 0.06 per cent; for ten patients, between 0.061 and 0.07 per cent; for one patient, between 0.07 and 0.08 per cent; for six patients, between 0.081 and 0.09 per cent, and for two patients, between 0.091 and 0.1 per cent. The higher values, as a rule, were obtained for patients over 65 years of age. Seven patients with trachoma, used as controls, had uric acid contents varying from 0.042 to 0.05 per cent. Thus it must be considered that forty-four of the sixty patients showed an elevation of the uric acid content of the blood, and hence this change in purine metabolism must be considered a possible factor in the production of cortical cataract.

S. R. GIFFORD.

Lids

TARSITIS SYPHILITICA. H. KHALIL, Brit. J. Ophth. **21:** 648 (Dec.) 1937.

Khalil reports a case of syphilitic tarsitis in a woman aged 65. She had suffered from enlarged glands in the axilla for three years, for which she underwent an operation. Glandular enlargement all over the body followed the operation. There was thickening of all four lids, with ptosis of both upper lids. There were no other ocular symptoms. The Wassermann reaction of the blood was 3 plus.

The author gives a detailed clinical and pathologic account of the disease.

The article is illustrated. W. ZENTMAYER.

Methods of Examination

THE MONOCHROMATIC LIGHT OF THE SODIUM VAPOR LAMP AS A SOURCE OF LIGHT IN OPHTHALMOSCOPY. H. SERR, Arch. f. Ophth. **137:** 636 (Oct.) 1937.

Because of the fact that yellow rays penetrate fog better than white (mixed) rays, sources emitting yellow rays are used more and more for the lighting of roads (headlights of automobiles). The sodium vapor lamp furnishes pure monochromatic (yellow) rays. With such a lamp as a source of light, interesting and important ophthalmoscopic observations can be made. In eyes with opacities of the media, yellow rays give an ophthalmoscopic picture which is brighter and contains more details than the picture obtained with white (mixed) rays. The yellow rays even penetrate the normal pigment epithelium and thus make visible the choroidal vessels and pathologic changes within the

choroid (hemorrhages). All blood vessels in the fundus appear black and stand out clearly against the yellowish gray fundus. Actual tears of the retina can be distinguished from mere thinnings.

P. C. KRONFELD.

Neurology

ESSSENTIAL NEURALGIA (?) OF THE TRIGEMINAL NERVE (OPHTHALMIC BRANCH) DUE TO BACILLARY TOXEMIA: TUBERCULIN THERAPY. C. CHARLIN, Ann. d'ocul. **174:** 588 (Sept.) 1937.

In a former communication by Charlin a case was reported in which the patient suffered for several months with tenacious neuralgia of the nasal nerve, which disappeared after a routine Mantoux test. It had not reappeared after a year and a half. In the months following the disappearance of the neuralgia the patient received a course of tuberculin treatment.

In the same communication there was reported a syndrome of the nasal nerve associated with ulcerative keratitis which had resisted all local and general treatment but showed a spectacular amelioration after a Mantoux test.

According to the author, there have been many cases of blepharitis, conjunctivitis and scrofulous keratitis in which the condition was uninfluenced by any local or general treatment but cleared up after injections of tuberculin. To clarify this statement, he gives in minute detail the treatment and course in such cases.

S. H. MCKEE.

HISTOLOGIC AND MICROBIOLOGIC STUDIES OF THE OPTIC NERVE IN CASES OF DEMENTIA PARALYTICA. A. BIFFIS, Ann. di ottal. e clin. ocul. **65:** 161 (March) 1937.

The literature is reviewed in regard to the finding of spirochetes in the central nervous system and especially in the visual pathways. The author employed the two methods of Jahnel on material obtained at autopsy from eleven patients who had had dementia paralytica. Spirochetes were found in the cerebral cortex in nine instances. They were found in the region just adjacent to the chiasm once. In only one instance were they identified in the optic nerve itself, in an area of softening and sclerotic connective tissue. The technical difficulties are emphasized, since in two instances intense impregnation with silver nitrate made it difficult to be sure that spirochetes were not present. The organisms in the cortex were found entirely in the gray matter and usually in areas of perivascular infiltration. The autopsy specimen in which spirochetes were found in the optic nerve was the only one showing histologic evidence of atrophy of the optic nerve. The patient from whom this specimen was removed had been inoculated with malaria, which had resulted in an exacerbation of general symptoms. Photomicrographs and a bibliography accompany the article.

S. R. GIFFORD.

Ocular Muscles

HEREDITARY PARALYSIS OF THE ABDUCENS NERVE. R. C. LAUGHLIN, Am. J. Ophth. **20:** 396 (April) 1937.

Laughlin reports a case of hereditary paralysis of the left external rectus muscle in a child whose mother exhibited a typical Duane syndrome. He briefly reviews the literature and gives the following summary:

"A case of hereditary paralysis of the abducens nerve is presented, in which five members in three generations of the same family were afflicted; two were examined. Ten other cases are reported from the literature. A statistical analysis of the data shows that in this condition a majority of females and of left eyes are affected."

W. S. REESE.

SQUINT AND HETEROPHORIA, WITH SPECIAL REFERENCE TO ORTHOPTIC TREATMENT. W. H. MCMULLEN, Brit. M. J. **2:** 1015 (Nov. 20) 1937.

McMullen first reviews the theories which have been advanced to explain concomitant strabismus. They are: (1) the muscular theory, (2) the accommodation theory and (3) the theory of defective fusion faculty. In addition, the influence of hereditary anisometropia, of nervous factors and of social conditions must be considered. In conclusion, the author says that no one of these theories alone can explain the disorder and that probably squint is a clinical condition due to a number of causes.

An interesting description of the development of orthoptic treatment is given. The author was one of the first to attempt treatment along the lines outlined by Worth, but he did not obtain satisfactory results and became skeptical as to its value. It is not stated whether with the new development of orthoptic treatment he has changed his mind.

ARNOLD KNAPP.

Operations

SIMULTANEOUS DOUBLE PARACENTESIS OF THE CORNEA FOR ISOLATION OF CICATRICIAL PROLAPSES OF THE IRIS. A. MARKOVIĆ, Klin. Monatsbl. f. Augenh. **98:** 187 (Feb.) 1937.

Denig suggested iridectomy on each side of the prolapse for the isolation of a cicatricial prolapse of the iris or on one side of the prolapse in cases in which a coloboma of the iris is present. Marković has constructed an instrument with which paracentesis can be performed simultaneously on the two sides of the prolapse. It consists of a double lance knife with nearly straight inner sides. The blades are connected by crossed handles and can be set closer together or farther apart by means of a screw, according to the width of the prolapse. The iris is excised on each side of the prolapse by means of an iris forceps. An iris hook may be employed in aphakic eyes.

K. L. STOLL.

Parasites

OPHTHALMOMYIASIS INTERNA: REPORT OF A CASE. I. DERER, Klin. Monatsbl. f. Augenh. **98:** 339 (March) 1937.

A boy aged 7 years had suffered from inflammation of his right eye for from six to seven weeks. It was caused by a larva of a fly, Wohlfartia magnifica (Sarkophaga), in the anterior chamber. The larva was about 7 mm. in length and 1 mm. thick and consisted of nine segments. After extraction, it coiled up spirally. The vision was greatly reduced, the intra-ocular tension was increased to 60 mm. (Schiötz) and the lens was subluxated upward and toward the nose, so that its lower margin appeared in the pupil. The eye became free from irritation soon after operation, and the vitreous cleared sufficiently for Derer to observe through the clear space below the lens that the disk was atrophic and that the retinal vessels were decidedly narrowed. Vision remained limited to perception and projection of light. The condition of the disk and retinal vessels led Derer to believe that the larva entered the eyeball by way of the blood stream. The sclera, which was intact in this case, would, however, have offered no obstacle to the entry of this kind of larva, which is known to pierce even sinews and bones.

K. L. STOLL.

Pharmacology

REACTION OF OCULAR TISSUES UNDER THE INFLUENCE OF CHEMICAL AND PHARMACOLOGIC SUBSTANCES INTRODUCED INTO THE ANTERIOR CHAMBER AND VITREOUS. M. CORRADO, Ann. di ottal. e clin. ocul. **64:** 145 (March); 520 (Aug.); 811 (Dec.) 1936; **65:** 361 (May) 1937.

Corrado presents an extensive introductory review of the literature on the use of chemical and pharmacologic substances for the treatment of intra-ocular disease. The purpose of his investigation was to accumulate evidence as to the effects of many substances in the eye, so that eventually some rationale may be established for their use. The substances used were of two types, chemical (hydriodic acid, tartaric acid, trinitrophenol, lactic acid, formic acid, acetic acid, hypertonic, isotonic and hypotonic solutions of sodium chloride, ammonia, hydrogen peroxide and alcohol) and pharmacologic (compounds of calcium and gold, mercuric chloride, mercuric iodide, electrocolloidal iron, sulfur, arsenic and iodine).

The method consisted mainly in the injection of increasingly larger amounts of the compound used into the vitreous of a rabbit until a definite reaction was observed clinically. A similar procedure was followed in the anterior chamber.

The author arrives at the following conclusions:

1. Ocular tissues react with decreasing violence to chemical substances in the following order: acids, alkalis, sodium chloride, hydrogen peroxide and alcohol. Of the pharmacologic substances, mercuric chloride and mercuric iodide cause the most violent reaction, followed in order by compounds of calcium, gold, bismuth, sulfur, arsenic, iodine and electrocolloids of iron and silver.

2. At first the tissues react only slightly, but later the reaction is more intense. Such reactions are definitely inflammatory and depend on the concentration of the substance rather than on its physiochemical nature.

3. Histologically, the reaction is of an intense but diffuse inflammatory nature. Rarely (as in the case of alcohol in the anterior chamber) does atrophy of tissue take place without an inflammatory reaction. No histologic change was noted after the use of sodium chloride (physiologic solution).

4. The anterior chamber can tolerate higher concentrations of the same substance than the vitreous.

In summary, all acids and alkalis in weak or strong solutions act as irritants, causing detrimental changes in the tissue. Of the pharmacologic group of substances, mercuric chloride, mercuric iodide, bismuth and neoarsphenamine produce even in weak concentration lesions that are harmful to the structure and function of the tissues. The best tolerated substances are the iodides (in the anterior chamber), physiologic solution of sodium chloride (in the anterior chamber and vitreous) and electrocolloids of silver and iron (in the anterior chamber and vitreous).

F. P. GUIDA.

Physiology

A STUDY OF THE COMMUNICATION AND DIRECTION OF FLOW BETWEEN CEREBROSPINAL FLUID AND OPTIC DISCS IN THE RAT. J. Q. GRIFFITH JR., W. A. JEFFERS, A. G. FEWELL and W. E. FRY, Am. J. Ophth. **20:** 457 (May) 1937.

Griffith, Jeffers, Fewell and Fry give the following summary of their experiments on adult albino rats:

"1. The passage of thorotrast from the cerebrospinal space to the optic disc regions has been demonstrated by the X-ray and by histologic examination.

"2. In 16 rats rendered hypertensive by previous intracisternal injection of kaolin, thorotrast injected intracisternally failed to appear under X-ray examination and was distinctly less evident histologically in the optic-nerve sheaths or disc regions.

"3. The evidence supports the view that the normal direction of fluid flow in the perineural spaces of the optic nerves is from the cerebrospinal space toward the optic discs.

"4. The bearing of this on the mechanism of papilledema is discussed."

W. S. REESE.

ANALYSIS OF THE SPONTANEOUS PULSATION OF THE RETINAL VESSELS. H. SERR, Arch. f. Ophth. **137:** 478 (Oct.) 1937.

A man aged 70 with unilateral glaucoma simplex and without signs of cardiovascular disease showed the same type of spontaneous venous pulsation in both eyes, to which when the tension of the glaucomatous eye rose above 36 a typical arterial pulsation was added. Serr photo-

graphed these pulsation phenomena with a special motion picture camera made for this purpose by Dr. Hartinger. The pictures allowed exact determination of the temporal relation between arterial and venous pulsation. The proximal end of the retinal veins collapsed during the systole, while the retinal arteries collapsed during the last third of the diastole. The collapse of the veins coincided with and exactly paralleled the pulsatory increase in intra-ocular pressure. Thus, it was proved that the pulsatory variation of the intra-ocular pressure caused the venous pulsation.

P. C. KRONFELD.

Retina and Optic Nerve

ANGIOGLIOSIS RETINAE WITH REPORT OF TWO CASES. I. CZUKRÁSZ, Brit. J. Ophth. **21**: 368 (July) 1937.

The clinical notes on two cases of angiogliosis of the retina are given. In each case the eye was studied histologically. In the first case the ganglion cells were destroyed. The nuclei of the inner nuclear layer and the cones and rods were partially necrotic, and glial tissue was abundant between the nerve and Müller's fibers and among the remaining desolate neural elements. The vessels were increased in size and number throughout the retina, and the perithelium was thickened with hyaline degeneration. A tumor 2 mm. in width was lying posterior to the ora serrata behind and connected with the retina. It was composed of hyaline degenerated tissue, cholesterol crystals and vessels. In the neoplasm were irregularly shaped spaces of various sizes, lined by endothelium and occasionally containing some blood. In some parts aneurysms were present.

In some sections at the ora serrata the retina was indefinitely divided into two layers. One layer was supposed to be of neuro-epithelial structure and the other of neural element. It was of interest that the tumor cells invaded the two primary structures of the retina, and the choroidal vessels.

In the second case a strange, reddish gray, dichotomic, worm-shaped formation was seen in the upper part of the globe between the retina and the choroid. The mass was growing to the temporal edge of the optic disk; it continued to the lower surface of the globe and extended like a fan onto the adjoining retina. The histopathologic picture of the retina was similar to that in the first case.

Czukrász surmises that in these two cases the neoplasm that was found, combined with the glaucoma, was responsible for the destruction of the retina. The tumors were classified as hamartoma of the same type as the dysontogenetic tumors of the central nervous system. They are of ectodermal origin, and the initiating cause lies in the neuro-epithelium.

The article is illustrated. W. ZENTMAYER.

RESULTS OBTAINED WITH SYMPATHECTOMY IN ATROPHY OF THE OPTIC NERVE AND PIGMENTARY DEGENERATION OF THE RETINA. F. BLOBNER, Klin. Monatsbl. f. Augenh. **98**: 289 (March) 1937.

Blobner discusses the idea of employing hyperemia therapeutically in diseases of the choroid, retina and optic nerve. Quoting investigators

in this field, Blobner refers to the results obtained with sympathectomy in atrophy of the optic nerve and in pigmentary degeneration of the retina. Magitot reported satisfactory results, which could not be substantiated by Löwenstein. Blobner reports the results obtained at the Eye Clinic of the University of Prague. Sympathectomy was done on thirteen male patients. Of these, six suffered from tabetic atrophy of the optic nerve; one had congenital syphilitic choroiditis associated with pale optic disks, and six had pigmentary degeneration of the retina. Eleven cases are described and illustrated with sketches of the fields of vision. The operation yielded no improvement in the six patients with tabetic atrophy of the optic nerve. Retardation of the pathologic process was only temporary in one of these patients, in whom the atrophy had advanced slowly for about five years prior to the operation.

The outcome was still more disappointing in the patients with pigmentary degeneration of the retina, in whom vision decreased and the field of vision became narrower after the operation. No difference in the volume of the retinal vessels could be noted with the ophthalmoscope. Disastrous complications, such as those reported by Abadie, Wilbrand, Kerr and others, did not occur.

Because of the result in these cases it was decided to abandon this none too harmless operation for the diseases just discussed.

K. L. Stoll.

Trachoma

Guide to the Treatment of Trachoma. E. Cornet, Rev. internat. du trachome **14:** 183, 1937.

There is no specific remedy for trachoma. Mechanical methods are of primary importance; chemicals are but adjuvant remedies and are especially valuable in the treatment of a secondary infection. The ends to be achieved are: evacuation of follicles, subsidence of papillary hypertrophy, resorption of pannus and cicatrization of ulcers.

J. E. Lebensohn.

Trachoma of the Fornix. E. Cornet, Rev. internat. du trachome **14:** 194, 1937.

Trachoma confined to the fornix is fairly frequent though difficult to detect except in the cicatricial stage. Since trachoma does not exist without involvement of the cul-de-sac, the latter must be considered the primary seat of the disease.

J. E. Lebensohn.

Healing of Trachoma. E. Olah, Klin. Monatsbl. f. Augenh. **98:** 350 (March) 1937.

Olah discusses the disputed question of the infectiousness of trachoma, which remains unsettled. It is safe to say that untreated trachoma is infectious whereas healed trachoma is not. A case to the contrary is reported. A man had trachoma twenty-five years before the present examination and had been considered cured by several oculists. Two years prior to observation of the patient by Olah, the right

eye became blind owing to a severe corneal ulcer, with remaining leukoma. A corneal ulcer developed in the left eye three weeks prior to the patient's admission to the clinic. At the time of admission the lower two thirds of the cornea was involved by a serpent ulcer. Separated from it, typical trachomatous pannus presented in the upper third of the cornea. The trachoma had been incompletely healed, in Olah's opinion, although it had become noninfectious, but had regained its infectiousness as soon as another infection occurred.

K. L. STOLL.

Uvea

CHRONIC TUBERCULOUS UVEITIS. H. D. LAMB, Am. J. Ophth. **20:** 490 (May) 1937.

Lamb reports a case of bilateral uveitis in a Negress in whom both eyes were eventually enucleated. He gives the following conclusions:

"This case is a typical example of chronic ocular tuberculosis as emphasized by F. H. Verhoeff. According to this writer, a condition is established in these cases in which the tissues of the host and the infecting organisms have become relatively tolerant to each other. The characteristic features in these cases are the following: Chronic ocular tuberculosis almost never occurs in cases in which there are well-marked clinical signs of systemic tuberculosis. Although chronic ocular tuberculosis is rare compared with the incidence of tuberculosis in general, yet when it occurs it is almost always sooner or later bilateral. It occurs almost exclusively in adults and far more frequently in females. The temperature is almost never elevated and is often subnormal. A large dose of tuberculin is usually required to produce a constitutional reaction and rarely a tuberculin reaction cannot be obtained at all. Animal inoculations of the lesions usually if not always give negative results."

W. S. REESE.

IS FUCH'S HETEROCHROMIA STILL TO BE CONSIDERED AS A MORBID ENTITY WITH ITS OWN PATHOGENESIS? J. STREIFF, Ann. di ottal. e clin. ocul. **65:** 298 (April) 1937.

In view of a recent report by Biozzi, who claimed that heterochromia is of tuberculous origin, Streiff refers to his previous extensive review on the question of heterochromia as a morbid entity with its own pathogenesis and discusses the subject. He concludes that true heterochromia must still be considered a morbid entity, which is related in etiology to disturbances in sympathetic innervation and to the status dystrophicus described by Passow.

S. R. GIFFORD.

Vision

THE RECOGNITION OF FLASHING COLOURED LIGHTS BY PERSONS WITH NORMAL AND DEFECTIVE COLOUR VISION. H. V. CORBETT and H. E. ROAF, Brit. J. Ophth. **21:** 592 (Nov.) 1937.

In testing for color vision it is important to vary the duration of exposure, as flashing lights and stationary lights viewed from a tossing vessel may be visible only for a short interval of time at each exposure.

The authors describe the lantern used and the methods of examination pursued. They give the following summary:

"Our results support those of Guttmann (1907, 1908) in showing that the person with defective colour vision is more dependent than the normal subject upon (*a*) the size of aperture, i. e., the angular aperture of a coloured light; (*b*) the brightness of the lights, and (*c*) the duration of exposure.

"It would seem that there are gradations in the degree of the defect so that one can recognize many degrees of defective colour vision. By the hypochromat red is more frequently named correctly than is green. This may be accounted for by the fact that the latter is less saturated or in other words it is not so far removed from parts of the spectrum which give rise to the blue sensation: the blue sensation being complementary to the second sensation of the dichromat (see Pitt, 1935)."

W. ZENTMAYER.

Sympathetic Ophthalmia

SUCCESSFUL DEMONSTRATIONS OF THE TUBERCLE BACILLUS IN THE STAINED SECTION OF AN EYE WITH SYMPATHETIC UVEITIS. J. MELLER, Ztschr. f. Augenh. **89:** 1 (April) 1936.

A woman 76 years of age had had glaucoma for four years. Physical examination gave essentially negative results for her age, except for a pleural adhesion in the apex of the left lung.

Iridencleisis was performed on the left eye without reducing the tension. One month later an extracapsular extraction for cataract was done on the right eye. Two weeks after operation severe uveitis was seen in both eyes. It did not subside in the right eye until after the left eye was enucleated, one month later.

The postoperative iridocyclitis was clinically characteristic of sympathetic ophthalmia. The greater part of the eye was used for tissue culture. This was found to be macroscopically positive for tuberculosis, and inoculation of an animal produced tuberculosis. Histologic examination of the remaining part of the eye showed only four small epithelioid nodules, which were typical of sympathetic ophthalmia. One was on the posterior surface of the iris, two were in the ciliary body and one larger one was in the choroid. The picture presented by the nodule on the choroid was also very similar to that seen in cases of tuberculosis without necrosis. The remainder of the choroid was scarcely altered. The section, stained after the method of Ziehl and Neelsen, showed tubercle bacilli in large numbers, located chiefly in the region of the pigment epithelium of the retina and the surrounding rods and cones.

In discussing this case, Meller points out many reasons for the negative tissue cultures and the difficulties in finding the bacilli in sections. Meller believes that if one accepts the similarity of the histologic pictures of sympathetic ophthalmia and tuberculosis, discussion as to whether the iridocyclitis in the eye operated on for cataract arose as the result of the operation on the left eye and was true sympathetic ophthalmia or whether it was a disease resulting primarily from the cataract operation is superfluous. He feels that sympathetic ophthalmia of the injured eye is nothing but a chronic (spontaneous) uveitis on a tuberculous basis.

H. GIFFORD JR.

Therapeutics

THE USE OF CONCENTRATED-EPINEPHRINE PREPARATIONS IN GLAUCOMA, IRITIS AND RELATED CONDITIONS. M. WIENER and B. Y. ALVIS, Am. J. Ophth. **20:** 497 (May) 1937.

Wiener and Alvis discuss the use of different preparations of epinephrine and give the following conclusions:

"1. Concentrated epinephrin is a powerful mydriatic capable of lowering ocular tension, or of producing an acute rise of tension, of causing severe pain, and occasionally unpleasant general symptoms; hence, the first use should be made where the patient can be observed for one or more hours.

"2. Epinephrin is a valuable aid in cases of chronic simple glaucoma in which miotics must be used for prolonged periods in lieu of the preferred surgical measures.

"3. Chronic secondary glaucoma generally does not yield to epinephrin but may do so, and a trial is justified.

"4. After operation for glaucoma, epinephrin may be effective in securing a state in which the tension remains normal for long periods without treatment.

"5. Glaucoma, after needling of cataracts and aftercataract membranes, has been controlled in a large percentage of cases by epinephrin with or without miotics.

"6. Epinephrin is invaluable in breaking up adhesions in cases of iritis and uveitis.

"7. The most convenient form for office and prescription use is as an ointment in a water-soluble base (tragacanth jelly) or in an oily base (petrolatum and lanolin)."

W. S. REESE.

Society Transactions

EDITED BY W. L. BENEDICT

AMERICAN OPHTHALMOLOGICAL SOCIETY

HARRY FRIEDENWALD, M.D., Baltimore, *President*

Seventy-Third Annual Meeting, Hot Springs, Va., June 3, 4 and 5, 1937

J. MILTON GRISCOM, M.D., Philadelphia, *Secretary*

BILATERAL METASTATIC CARCINOMA OF THE CHOROID: REPORT OF A CASE. DR. MARTIN COHEN, New York.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 604.

PRECANCEROUS MELANOSIS AND DIFFUSE MALIGNANT MELANOMA OF THE CONJUNCTIVA. DR. ALGERNON B. REESE, New York.

This paper will be published in full, with the discussion, in a later issue of the ARCHIVES.

MELANOMA OF THE IRIS: REPORT OF A CASE. DR. FRED T. TOOKE, Montreal, Quebec.

A pigmented spot in the iris of the right eye had been observed for many years and had not increased in size. More recently it had become definitely larger, and the question of malignancy was raised. Biopsy showed no evidence of malignancy, and there was no recurrence after nine months.

DISCUSSION ON PAPERS BY DRS. COHEN AND TOOKE

DR. WILLIAM ZENTMAYER, Philadelphia: I should like to report a case in connection with Dr. Tooke's paper. In February 1932 a woman aged 27 consulted me because of a growth on her right eye. She stated that from birth she had had a dark spot on the iris of this eye and that it had remained unchanged until two months previous to her visit to me, when it grew rapidly. It then involved one sixth of the circumference of the iris, extending from the pupillary border to the filtration angle, and was slightly elevated and somewhat irregular in contour. Vision in the right eye was 6/6 and in the left eye, 6/25. I succeeded in extirpating the growth in toto, together with some of the normal structure of the iris on each side. At a recent examination there were no signs of a recurrence of the growth. Another interesting feature in connection with this case is that the amblyopic eye, which had a corrected vision of 6/25, now has a vision of 6/9.

The difference between my case and that of Dr. Tooke is that in my case the growth assumed a malignant character. It proved to be a melanotic sarcoma.

Dr. John W. Burke, Washington, D. C.: I wish to report a case similar to that of Dr. Cohen. My patient was followed from the incipiency of the metastasis to death.

Mrs. V. M. V., aged 49, was first seen at my office in February 1929. The vision in each eye was 6/4.5. The fundi were normal. She was seen again in March 1931, with vision of 6/4.5 in each eye and normal fundi. On Feb. 15, 1932, I was called to see her, as she had been in bed for a week and had been treated for nervousness by her family doctor. While I was taking her history, she remarked that for several days her vision had been blurred and that she had had subjective light flashes constantly. Externally, the eyes were normal, and no visual tests were made in her home.

On examination of the fundi a grayish mass was seen in each macular region. The mass in the right eye was about 2 disk diameters, and that in the left eye was slightly larger. The mass in the right eye was elevated 4 diopters, and that in the left eye, 5 diopters. The process was entirely subretinal in the right eye; the edges were not clearly defined and were irregular in outline, but the remainder of the fundus was normal. In the left eye there was a small amount of exudate in or on the retina, at the apex of the mass. There were no opacities of the vitreous, and no hemorrhage was seen at any time in the progress of the disease. The tension was normal. The patient remained in the Episcopal Hospital for three weeks. A survey was made, but nothing was found except one infected tooth, which was removed.

During the patient's stay in the hospital there was no change in the fundi. A second physical examination was made. At this time a small lump, about the size of an English walnut, was found close to the wall of the chest, at the upper part of the breast, which had not been detected at the first examination.

The patient was under periodic observation during April, with no particular change occurring in the eyes. Her vision, taken somewhat roughly in her apartment, was 6/50 in each eye. Her subjective symptom of light flashes remained unchanged. Early in April she called attention to the presence of lumps in the upper part of her left arm, in the postcervical region on the left side, under the left breast and in the postcervical region on the right side. These growths did not enlarge after their initial appearance. On May 1 a detachment of the retina was found in both eyes.

The patient died suddenly on May 20. Autopsy disclosed the following: (1) scirrhous carcinoma of the breast and (2) metastatic adenocarcinoma of the choroid, lungs, liver, pancreas and adrenal glands. Microscopic examination of the left eye showed slight edema of the iris and of the tissues at the filtration angle. On the temporal side of the section the choroid was thickened by infiltration with adenocarcinoma having a sclerosing stroma. There was some degeneration of the pigment epithelium of the retina, and a small amount of serous exudate between the retina and the choroid was present. The infiltration extended approximately to the equator and had advanced out along the sheath of the posterior ciliary artery. The right eye was essentially the same as the left.

Dr. Ernest F. Krug, New York: I should like to add a few words to Dr. Cohen's report. That these cases are rare is true. In over twenty-

five years' clinical experience I have seen only 2 cases, and their similarity was marked; they were both in women under 40 years of age who had a history of primary involvement of the breast and eventually involvement of the lung. The patient in whom the condition developed the most recently is still alive. This case is particularly interesting since metastasis developed in the other eye. I removed the right eye in October 1936. The breast had been removed in 1934. The appearance of the mass in the right eye coincided with the recurrence in the amputation scar.

CAUSES OF SENILE CATARACT. DR. EDWARD JACKSON, Denver.

Certain causes of cataract are known, such as mechanical damage to the lens or diabetes. Other conditions are often suspected of causing cataract, and probably still others have not been considered in this connection. The term "senile" is appropriate because it gives a long period for many possible causes to operate. A comparison of experience should enable one to judge which of the suspected and unsuspected causes are probably responsible for the impairment of vision.

DISCUSSION

DR. C. A. CLAPP, Baltimore: I should like to ask Dr. Jackson one question. If the loss of water is the cause of the lenticular opacities, why is it that cataract does not develop in boiler inspectors, who are subject to high temperatures and who lose a large amount of water by perspiration but who are not exposed to light rays?

DR. EDWARD JACKSON, Denver: With reference to Dr. Clapp's question, I should say that further investigation on the subject is necessary.

I feel that there are not now enough facts to enable one to arrive at any conclusions. It may be that a cataract represents a deviation from normal nutrition in relatively recent structures. The human crystalline lens is definitely a more highly elaborated organ than the lens of any of the near lower animals, and it is entirely different from the lens of the invertebrate animals.

THE EYE AND DIABETES. DR. HANS BARKAN and DR. HORACE GRAY (by invitation), San Francisco.

The varied terminology used by ophthalmologists to designate abnormalities of the fundus in cases of diabetes is confusing to the internist who is treating the patient. On the basis of a clarified terminology, an attempt should be made to estimate from the enormously varying statistics in the literature the true probable frequency of the various ocular findings in patients with diabetes. Until comparable statistics on the results of treatment are available, one must depend on clinical impressions for the assessment of the value of any treatment as regards the eye in cases of diabetes.

DISCUSSION

DR. C. A. CLAPP, Baltimore: In discussing Dr. Barkan and Dr. Gray's paper, I should like to show a slide to illustrate the theory of

Waite and Beetham that retinitis starts first as a hemorrhagic condition. Retinitis is rarely found in the young. The condition shown here, in which there is no evidence of exudation, occurred in a youth aged 19. I feel that if persons with retinitis were observed in the early stages hemorrhages would be noted first and the exudation later. After these hemorrhages are seen, several years may elapse before exudation occurs. I believe that there is a definite diabetic retinitis, because one can make a tentative diagnosis of a diabetic condition in a person with retinal involvement before the general physical examination has been made, which usually corroborates the ophthalmic diagnosis.

DR. HANS BARKAN, San Francisco: In general, Dr. Gray and I have found that the statistics and the observations of Waite and Beetham and those of Braun and Mylius agree quite well with each other and seem to bear out certain points which we are presenting here. If one looks at the illustrations of diabetic retinitis in "Kurzes Handbuch der Ophthalmologie" one finds a plate illustrating diabetic retinitis which is an absurdity. It might as well be a picture of myelogenous leukemia or of bacterial endocarditis. It is not typical of the retinitis found in patients in the early stages of diabetes. We want to stress the point that there is such a picture and that one can tell in the majority of cases that one is dealing with diabetes primarily and not with arteriosclerosis or with hypertension associated with arteriosclerosis or with albuminuric retinitis.

DETACHMENT OF THE RETINA: OPERATIVE RESULTS IN ONE HUNDRED AND SIXTY-FOUR CASES. DR. JOHN H. DUNNINGTON and DR. JOHN P. MACNIE (by invitation), New York.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 532.

ETIOLOGY OF IDIOPATHIC RETINAL DETACHMENT. DR. ALEXANDER E. MACDONALD, Toronto, Canada.

An explanation of the etiology of idiopathic retinal detachment based on hydrodynamic grounds is suggested.

DISCUSSION ON PAPERS BY DRS. DUNNINGTON AND MACNIE AND DR. MACDONALD

DR. E. V. L. BROWN, Chicago: Acute edema as a cause of retinal detachment was the subject of an extensive paper by Krückmann at the 1936 meeting of the German Ophthalmological Society at Heidelberg. He elaborated on the mechanism, basing his remarks on a study of cases in youths aged 18, 20 and 25, respectively, who were carrying heavy loads when detachment occurred. They were bent over and going up a grade. In each case the load suddenly slipped, the head was thrown up from the lowered position, the eyes turned up further at the same time, and a pull on the inferior oblique muscles resulted. In each patient a detachment of the lower temporal quadrant of the retina was found the next day. Krückmann described the retinas as having the same ground-glass color as is seen in cases of Berlin's edema, namely, a peculiar milk-glass appearance. Krückmann asserted that when the eyes make a

sudden movement upward there is marked abduction of each eye. He expressed the belief that the lower temporal portion of the sclera is pulled away by the inferior oblique muscle from its position against the structures within the eye and that ischemia, and then edema, of the retinal vessels are produced. He also gave extended consideration to ischemia in relation to edema of the brain. He stated that his 3 cases of sudden retinal detachment which occurred while the patients were carrying heavy loads are unique, but the 5 cases reported here indicate that such cases may be frequent.

DR. LUTHER C. PETER, Philadelphia: To the ophthalmologist every phase of retinal separation is important. The patient, however, is interested in only one phase, and the questions which he naturally asks are: What are my prospects of recovery? How much vision will I have?

I believe that the prognosis should be made clear to the patient as to the possibility of reattachment, with the prevention of all the complications which follow retinal separation, and as to the possibility of restoration of central vision. Central vision, in my experience, is influenced (1) by the duration of the detachment and (2) by the proximity of the tear to the macula. This statement does not differ in any way from the report which Dr. Dunnington and Dr. Macnie made on this point, excepting as to the matter of time. I mean by this that a separation of three months' duration or longer is likely to leave some macular disturbance. As a matter of fact, retinal sensitivity, as has been pointed out, is reduced in practically every case, but macular vision may suffer, even in cases of recent detachment, although fairly good central vision can be obtained in those cases in which the detachment is of no longer than three months' duration. As has been pointed out, the prognosis in general is influenced to a large extent by the etiologic factors.

From the standpoint of surgery, although this phase of the subject has not been gone into in any detail, I believe there are three things which should be considered in conjunction with this comprehensive report. The first is the thoroughness of the preliminary study. It seems best to place the patient in the hospital for this purpose, even for a week before operation is attempted, during which time careful measurements can be made and recorded.

The second point is that of sealing the tear. I have found, in my later experience, that it is unnecessary to create many points of adhesion between the choroid and the retina; in other words, if the tear is surrounded by a proper number of needle punctures, which are carried out to the periphery and along the ora serrata, one can avoid a great deal of surgical trauma, which perhaps used to be more extensive than it is at present because the operator wanted to be sure to seal up every part of the retina that might cause a recurrence of the separation.

The last point which I feel is important is that of drainage. If the drainage is adequate and the tear has been properly sealed, the results should be good, and the percentage of good results will continue to increase. I am now using a small trephine, about 1.5 mm. in diameter, so as to be sure to obtain adequate drainage. It also reduces the number of perforations with the needle point, because one can use the Larssen

method to a considerable extent; this method is less damaging, and hemorrhages into the vitreous are naturally not so likely to occur.

Dr. Edward N. DeWitt, Bridgeport, Conn.: Dr. Dunnington and Dr. Macnie are to be congratulated on the remarkable success they have had in replacing detached retinas. I have found four distinct types of retinal detachment with which poor results are obtained: (1) detachment of long standing, with many tears and low intra-ocular tension; (2) detachment associated with circulatory disturbances, such as essential or malignant hypertension and arteriosclerosis, and with multiple hemorrhages in the retina and papilledema; (3) detachment associated with nephrosclerosis and, to a lesser extent, with diabetes, and (4) detachment resulting from scar formation; that is, detachment resulting after removal of a foreign body from the retina months or years previously. Such detachments are due to contraction of scar tissue in the retina, and even if replaced by operation the retina again becomes detached a few months later.

Dr. E. C. Ellett, Memphis, Tenn.: I should like to ask Dr. Dunnington to discuss detachment of the retina in aphakic eyes.

It is comforting to hear the experience of others on eyes without retinal tears, but my impression is that the most satisfactory results have been obtained in eyes with accessible tears.

Gonin's early reports were too sketchy to enable one to understand his technic, and in my anxiety to do something I treated 2 patients with injections of mercuric cyanide, after the Surdille method. Both attempts resulted in failure. When I had learned more of Gonin's method, I procured a suitable cautery and did a number of cautery operations, with some success. The contact diathermy method of Larssen, in use at the time at Moorfields, appealed to me and is still my favorite method. For this method I use the Gradle needle, which partly perforates the sclera, and then surround the tear, if any, with Walker's micropins and trephine the sclera. In the meantime I tried Guist's potassium hydroxide method, but was not impressed by it.

My experience is not the same as that of Dr. Dunnington and Dr. Macnie, namely, that "the duration of the detachment had comparatively little effect on the operative result." I feel that recent detachments are the most amenable to operative procedure.

My total number of operations to date is between 50 and 60 on 48 patients. I have twice operated on a patient with unrecognized tumor of the choroid, and I can offset that experience by reporting cure of a detachment in a case in which tumor had previously been diagnosed. I have had several patients, indeed, a number, in whom an anatomic cure resulted; that is, the retina was replaced, but restoration of function was disappointing. The field of vision, but not the visual acuity, was fully restored. In some patients a macular lesion was seen, which most often appeared as a hole, but in others no definite cause for the poor vision could be found.

Since the prognosis of untreated retinal detachment remains almost 100 per cent unfavorable, I feel that every patient should be given the benefit of operation.

The arbitrary confinement to bed for one or two weeks in as near an immobile position as possible seems often unnecessary and at most is merely a guess at what is necessary. If the attempt to produce an

adhesive inflammation is successful, it would seem, judging from the behavior of other wounds not subjected to tension, that a shorter period of immobility would suffice, especially since the immobility is at best only relative.

DR. ARNOLD KNAPP, New York: I was particularly interested in a statement in Dr. Dunnington and Dr. Macnie's paper to the effect that their results in eyes without retinal holes, or their results with "blind" operations, were better than the results in eyes with retinal holes. I am sorry to say that my records do not bear this out. I have the detailed histories of 78 cases in which I have operated; holes were found in 68, varying in number from 1 to 12 in a single case. There were no holes in 10 cases. The results in these 78 cases consisted of cure in 53, or 68 per cent, and improvement in 6; if the results in these two groups are considered together, cure was obtained in 75 per cent. There were 19 cases, or 25 per cent, in which there was no improvement. The cases of aphakia have presented the greatest difficulty. I have had successful operative results in only 1 of 4 such cases. In the 3 in which operation accomplished nothing, no holes could be found. In the case in which operation was successful, a hole was found, which was closed.

I do not believe that it is so much the extent of the detachment as the size of the hole that is important; the giant holes in particular are difficult to treat. Those in the ora serrata do well. The cases in which holes have not been present have, in my experience, been cases of aphakia and of choroiditis.

It seems to me that one objection to blind coagulation is that too extensive damage is done to the eyeground. Not enough is known as to how much coagulation the eye will stand, but it surely is ideal if a hole is present simply to close it and then do a drainage puncture if the condition demands it.

The reason why the results in patients with low tension are poor is that the vitreous is disorganized or fluid. However, in some of these patients something can be accomplished by surface coagulation.

One great difficulty in operations for detachment remains—macular involvement. It is still not understood why, after a comparatively good result so far as replacing the retina is concerned, a macular change which impairs the visual result should develop.

DR. CLIFFORD B. WALKER, Los Angeles: I have been particularly interested in these two excellent papers. With regard to the occurrence of bilateral retinal detachment, as recorded by Dr. Dunnington and Dr. Macnie, I should like to ask whether in their series there were any instances of simultaneous bilateral detachment resulting from the same injury. None has appeared in 250 patients whom I have examined, although I know of an instance caused by the patient sitting down very hard on the floor when a chair was jerked from under him. In the literature these cases are few, almost rare.

Why does retinal separation occur, as a rule, in one eye only, although the injury often apparently shocks both eyes quite equally? Because of this fact it is usually argued that the retina which separates must have been degenerated more than the other, although the ophthalmoscope often does not confirm this. This may be true in some cases, but I do not believe that it accounts for the frequency with which tears occur in the upper temporal quadrant of the retina in the neigh-

borhood of the attachment of the superior oblique muscle. Here, at the top of the globe I believe, the retina is under additional mechanical disadvantage in the presence of the peculiar punishment of the powerful mechanism of pulley, long tendon-short contact arc.

To throw further suspicion on one superior oblique muscle, one must consider the conjugate positions of the eyes, wherein only one eye either is being acted on most strongly by its superior oblique muscle or stands in a position for decided or peculiar punishment by its superior oblique muscle. Furthermore, the long (19 mm.) tendon of the superior oblique muscle, after descending at an angle of 15 degrees, has its center of attachment from 3 to 4 mm. beyond the superior pole of the globe, where the sluggish weight of the ocular media helps a radical jerk on the sclera to produce a partial vacuum between the vitreous and the sclera. Diminishing waves of pressure and vacuum may follow the jerk, but this type of punishment is just the sort to cause a cystically degenerated or otherwise weakened area to form the original tiny hole that leads to the formation of a tear. The natural lack of contact arc of the superior oblique muscle is ameliorated less and less by the overlapping superior rectus muscle as the eye turns downward and outward.

Although the superior rectus muscle is relaxed as the eye turns downward, and uncovers the superior oblique tendon more and more as the eye turns outward, still it smothers most of the radial or centrifugal component of the pull of the superior oblique muscle during the first 15 or 20 degrees (fixation) of scleral movement toward the pulley. My measurements, models and diagrams show that the pulley center is about 20.5 degrees away from a point 1.25 mm. back of the center of the eye. If the center point of attachment of the superior oblique muscle is moved 13.25 mm. by rotation (looking downward and outward) until it reaches the 20.5 mm. line, the pull of the superior oblique muscle becomes largely radial or lifting on the sclera in spite of the relaxed superior rectus muscle. At 9.5 mm. of rotation (45 degrees of fixation downward and outward) the superior oblique muscle would have to lift only the nearest border of the limp superior rectus muscle about 2.5 mm. more than in an ordinary rotation in order to develop a lifting centrifugal component to the sclera of about two thirds of its total pull at that time.

An added stimulus while the eyes are at, or half-way toward, this critical conjugate position could, I believe, damage only the retina of the eye of the side to which the gaze was directed, even though both fourth nerves were equally affected by such injuries to the spine, cranium or body as could produce longitudinal transmission to the mesencephalic region. If other nerves were stimulated at the same time, tending to jerk the eyes back into normal position or farther, while the eyes were still being held in conjugate deviation, the action of opponents could give a similar result by jerking the *eye away* from the still taut and tethering superior oblique tendon.

This explanation is the most satisfactory one I can give to account for the "one side only" phenomenon, if it is possible for the stimulations to originate in some such manner as stipulated. Certainly an ocular muscle is strong enough (from 500 to 1,000 Gm. pull) to give the sclera a vicious jerk.

The softer the eye, the more effective this mechanism can be. I therefore advise and urge all patients with low ocular tension to wear pupil goggles and avoid hazards carefully if they really wish to protect their vision. I am especially insistent on this in cases of aphakia before and after operation. Detachment of the retina in cases of aphakia is so hard to cure that I believe more "cures" will really be obtained with strict enforcement of "pupil goggle prevention" the first year after operation, if tension is low, than with the present methods of surgical treatment. The postoperative aphakic eye continues to gain in strength for more than a year. Many patients will not be treated for a whole year, but I am sure that every possible immobilization treatment is helpful, in spite of the fact that in this matter Vogt does not follow Lindner, to whom credit is due for pupil goggles.

In separations primary in the lower part of the retina, without a visible tear, as analyzed by Dr. MacDonald, it must be remembered that an invisible single broken cyst in the periphery can give rise to a separation similar to a pinhole in a tire, and yet the tear may remain small and hidden because the weight of the vitreous inhibits such mechanics as occur in the upper polar region and the inferior oblique muscle has no such punishing ability as the superior oblique muscle. Krückmann's complicated analysis of the mechanism of retinal detachment, as pointed out by Dr. Brown, must also be considered in this connection. Further, the phylogenetic youth and weakness of the inferior portion of the retina and macula must not be ignored. In general, on the basis of my experience I believe that detachments in the lower portion of the retina, with or without visible central or peripheral holes, are quite amenable to treatment in persons up to 40 years of age with or without myopia.

Dr. Eugene M. Blake, New Haven, Conn.: Dr. MacDonald's paper is interesting and proves that detachment can occur as a result of lifting heavy weights. It gives one something to refer to. A year ago I saw a man of 29, whose occupation involved the lifting of heavy bundles of paper, in whom detachment of the retina occurred. The question arose as to whether or not this injury was compensable. It seemed to me reasonable to attribute the detachment to his occupation. Operation resulted in complete reattachment.

Dr. Sanford R. Gifford, Chicago: No attention has been called to cases in which a hole occurs in the upper portion of the retina while the detachment occurs in the lower portion. I have seen a number of cases in which this occurred; such cases seem to prove the effect of the tear in the production of detachment more than anything else. In the first case in which I recall operating I was afraid not to cauterize below, so I cauterized in the region of the hole and below and obtained a good result, but in 2 later cases I did not do anything below the hole except trephination. I cauterized in the region of the hole and was able to close it, but I did nothing below the hole except to let the fluid out. Such cases are common and are responsible for some of the reports in which it is stated that holes were not found.

I wish to call attention to the value of the Friedenwald ophthalmoscope in discovering holes. I have found in cases in which one hole was discovered with the ordinary ophthalmoscope with very good light

that four or five holes could be found with the fine beam of the Friedenwald ophthalmoscope. Also, in certain cases in which holes are suspected the fine beam enables one to decide whether or not the hole is complete, as one can see the edges so entirely that there can be no doubt about it.

DR. JOHN H. DUNNINGTON: There are a few points which I should like to add. Trephining to insure free drainage of the subretinal fluid was used in a considerable number of these cases, but a study of these did not lead to any conclusion as to its efficacy. It seemed to us that sufficient drainage was obtained from the punctures made with the Walker needles or with the electrodiaphake of Lacarrère.

I cannot answer Dr. Ellett's question as to why the prognosis is so unfavorable in the cases of aphakia. The study of 13 cases did not lead us to any definite conclusion along this line. In those cases of retinal detachment associated with aphakia, the latter was present before the detachment had its onset.

We do not wish to give the impression that the duration of the detachment is of no importance, but we hoped rather to encourage treatment in cases of long standing.

I also cannot answer Dr. Ellett's question regarding the length of the period of confinement in bed; i. e., whether it is necessary to keep the patient there as long as most ophthalmologists do. I really believe that he is correct and that it would be safe to let the patient up much earlier, but we have followed the usual teaching.

In regard to the incidence of bilateral detachment which Dr. Walker spoke of, I am afraid that we did not make ourselves clear. In the paper we simply mentioned that detachment of both eyes occurred in 6 per cent of the cases. We did not mean that they had occurred simultaneously, for often a period of several years elapsed between the detachment in one eye and that in the other. Our only reason for mentioning it was to call attention to the frequency with which bilateral detachment does occur, in the hope that this knowledge might serve as an additional stimulus to all ophthalmologists to conserve all possible vision in every case of retinal detachment.

DR. ALEXANDER E. MACDONALD: I wish to thank Dr. Brown and Dr. Walker for calling my attention to Krückmann's paper. I did not know of it previously. I must say that I am not particularly impressed by the possibility of the action of the oblique muscles causing detachment. It seems to me that the synkinesis of voluntary movement is not so rapid as one finds in nystagmus or in nystagmoid jerking, and I am not aware of any case in which detachment of the retina is associated with these conditions.

If my work is confirmed, it is important to pay attention to the after-treatment of retinal detachment. Patients should avoid all lifting strain, and even bending down should be done with care.

I wish to call attention to the high levels of cerebrospinal fluid pressure that may be found in metabolic diseases. If such cases are followed more carefully, a definite relationship may be found; that is, the intensity of the retinal changes may go hand in hand with the level of the cerebrospinal fluid pressure.

NEUROMYELITIS OPTICA: A REPORT OF TWO CASES. DR. HANFORD McKEE and DR. FRANCIS McNAUGHTON (by invitation), Montreal, Canada.

The first clinicopathologic studies made on neuromyelitis optica by Achard and Guinon in 1889 and the later studies made by Devic in 1894 are cited. Since 1894 the condition has been referred to as Devic's syndrome. The striking symptoms observed in 2 typical cases seen at the Montreal General Hospital are described.

DISCUSSION

DR. SANFORD R. GIFFORD, Chicago: I should like to place on record 2 cases which I consider to belong definitely to this syndrome, and 1 case which I believe is of similar nature. Of the first 2 cases, 1 was reported by Dr. Perritt from the Cook County Hospital in 1934, at which time he mentioned the other case. This condition is so dramatic that it could never be overlooked if a typical case of it has been seen before.

The first case was that of a boy of 12 years. When I saw him the disk was elevated 6 diopters, and there was no light perception in the affected eye. The other eye was normal. The boy was perfectly healthy. In this case one could think only of a tumor in the optic nerve. The condition was not diagnosed. Two weeks after I saw him, he fell while walking and was taken to the hospital, where a diagnosis of transverse myelitis was made. During his stay in the hospital the vision in the other eye decreased to perception of hand movements; no change in the disk was observed at any time. In the eye first affected typical optic neuritis was present; in the second eye there was retrobulbar neuritis. In two months the condition had gradually cleared up, and two months later the vision was 20/20 in each eye. The nerve of the first eye was pale and showed changes resembling secondary atrophy, but vision was normal. In the other eye there was a slight pallor of the disk.

The second case, reported by Perritt, was that of a girl of 5 years. In this case myelitis had preceded optic neuritis. The child had had measles three or four weeks before, with paralysis of one leg. One day when she awakened she could see nothing with either eye. She was brought to the hospital, and mild optic neuritis was found in one eye. There was no light perception in either eye. The condition cleared up later, and she left for another city; reports from there showed vision of 20/20 in one eye and 20/40 in the other. Examination of spinal fluid showed 97 cells per cubic millimeter.

Recently Dr. Lawson, of Evanston, Ill., and I observed a girl aged 9 years whose symptoms were confined to a sudden loss of vision first in one eye and then in the other. There was mild optic neuritis in both eyes, and the vision was 5/200 in each eye, with a large central scotoma. Spinal puncture and neurologic examination gave negative results. No myelitis was present, and vision has returned practically to normal. Dr. Lawson has not mentioned any elevation of the disks at the present time.

An interesting question has been raised as to whether the changes of multiple sclerosis will develop later in these cases, because changes occurred in a few weeks which in cases of multiple sclerosis occur during the course of years, and it has been suggested that these represent an

acute form of sclerosis. I do not know of any cases in which the patients have been followed long enough to determine whether later signs of multiple sclerosis will develop.

Dr. Alan C. Woods, Baltimore: I have been so unfortunate as to see a number of cases of neuromyelitis optica and cases of Schilder's disease, encephalitis periaxialis diffusa, all of which constitute apparently the same pathologic condition—an acute demyelinating disease with different clinical syndromes. As Dr. Gifford and Drs. McKee and McNaughton have emphasized, at the onset these conditions are often quite similar. The ocular symptoms predominate, and there may be great confusion as to the exact diagnosis, which becomes evident only with the later development of the myelitis. Owing to the early diagnostic uncertainty, 3 of these patients were subjected to operative procedures—1 to an exploratory craniotomy, 1 to an injection of the ventricles with air and 1 to the opening of the posterior nasal sinuses. The ultimate visual outcome in all these patients was bad. Those patients not operated on and not subjected to a lumbar puncture made remarkable recoveries. In these dramatic cases it seems to me that any operative or traumatizing procedure, even a lumbar puncture, is contraindicated.

The Possibility of Möller's Muscle Acting as an Opponent. Dr. Clifford B. Walker, Los Angeles.

After construction of a large model of the orbit, with the eyeball and extra-ocular muscles in the primary position, it has been demonstrated that a resultant force from these muscles could be active, at least by tonicity, tending to pull the eye toward the nasal wall. Müller's muscle, fortifying strongly developed layers of Tenon's capsule, is considered from the functional standpoint of oppositional action to such a nasal resultant rather than as a purely vestigial remnant.

Divergence Insufficiency: A Clinical Study. Dr. Avery D. Prangen and Dr. Ferdinand Koch (by invitation), Rochester, Minn.

Divergence insufficiency is characterized by esophoria in both distant and near vision; esophoria is greater in distant vision. Abduction is persistently below normal, and homonymous diplopia is elicited easily on fatigue or on breaking up of binocular fixation. Although the anomaly is one of divergence, symptoms are more marked in near vision. The condition occurs in tense persons who are engaged in the types of higher occupation which require much close work. Treatment by means of a full balanced correction of the existing ametropia and anisometropia, together with prism base out, is suggested. An early or even premature correction for presbyopia also seems to be of benefit.

DISCUSSION

Dr. James Watson White, New York: I have not found, as Dr. Prangen has, that the symptoms of divergence insufficiency are more marked at the near point; rather the panorama asthenopia and double vision are much more annoying for distant vision. There is always an

esophoria or esotropia for distant vision, but at 33 cm. there may be only a trace of esophoria. Frequently there is an exophoria at 25 to 30 cm., and at 20 cm. there is practically always an exophoria. The convergence near point is rarely excessive and is more likely to be remote. The esophoria or esotropia, as well as the diplopia for distant vision grows progressively less as the light approaches the eyes, and both disappear at some point within 1 meter, depending on the amount of the deviation. It is an interesting observation, although usually not necessary to a diagnosis, to measure the amount of the esophoria and also of the diplopia, at 6 meters, 3 meters, 1 meter, 0.5 meter and 0.25 meter. In doubtful cases the deviation for distant vision should be measured at 25 to 50 meters also. The test for diplopia is not so dependable as the actual measurement of the amount of deviation by the screen test.

I have found, as Dr. Prangen has, that there is some degree of hypocyclosis for which a plus lens must be added for reading. This plus lens, however, does not reduce the esophoria for distant vision if the esophoria is due to a divergence insufficiency. However, if the esophoria is due to a convergence excess, the esophoria for near vision is definitely lessened. If the esophoria is about the same for distant as for near vision and if atropine or plus lenses will definitely reduce it, the condition is probably a convergence excess; but if the esophoria for near vision is considerably reduced, or if it is changed to an exophoria, and the esophoria for distant vision is practically unchanged, then a primary divergence insufficiency probably exists.

It is possible that some of the patients observed by Dr. Prangen primarily had convergence excess. This would seem probable, since the symptoms for near vision instead of for distant vision were so much increased, as in a typical divergence insufficiency.

Prism divergence for distant vision is below normal and may be nil, whereas for near vision the prism divergence may be quite normal. I have not found a prism divergence of as much as 3 to 4 diopters, as Dr. Prangen has.

Prisms base out in glasses for distant vision are well borne, but if these glasses are used for close work the symptoms may increase. The prisms in the reading correction tend to act as a prism convergence exercise and to increase the esophoria for near vision. This is proved by the fact that if the patient is using prisms base out when first seen, removal of the prisms will not change the esophoria for distant vision, whereas the esophoria for near vision will be definitely lessened.

No form of divergence exercises has been of any aid in my cases of divergence insufficiency. However, if the condition is one of convergence excess, divergence exercises are a definite aid. I have not found the high incidence of nervous symptoms and constitutional conditions which Dr. Prangen has found.

DR. THOMAS D. ALLEN, Chicago: I think one of the tests which Dr. Prangen did not have time to speak about should be emphasized, and that is the test of the patient's ability to fuse. If a person obviously has no ability to fuse, prisms will do no good. Occasionally prisms may be of some use when there is weak fusional ability. I usually test for stereopsis and I presume Dr. Prangen always does. I find when I am a little hesitant about fitting prisms that it is of great value to put into a

lightweight frame the glasses that I believe should be used and then to add or subtract the prisms and let the patient wear the glasses for a half-hour or longer in the office.

RELATIONSHIP OF HETEROPHORIA TO DIVERGENCE AND CONVERGENCE BASED ON CLINICAL MEASUREMENTS. DR. F. H. HAESSLER, Milwaukee.

An attempt is made to help elucidate one of the fundamental concepts of heterophoria; namely, how it is related to the functions of convergence and divergence. For this purpose, clinical measurements of heterophoria, convergence and divergence on 1,000 patients were analyzed and presented.

SURGICAL MANAGEMENT OF PTOSIS, WITH SPECIAL REFERENCE TO THE USE OF THE SUPERIOR RECTUS MUSCLE. DR. P. CHALMERS JAMESON, Brooklyn.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 547.

DISCUSSION

DR. SANFORD R. GIFFORD, Chicago: I wish to mention another operation for ptosis, the value of which I have discovered in the last two years, and that is the Blaskovics operation, as modified by Lindner. I want to show the typical results of this operation in 2 cases.

The first case was one of typical bilateral ptosis. The Blaskovics operation is a resection of the levator muscle. I did not perform this for a long time because I did not believe it applicable to cases of complete ptosis until Lindner showed that it was. By resecting the tarsus and making the advancement of the muscular tissue, which is always present if it has no apparent innervation, good results are obtained, even in cases of complete ptosis.

The second case was that of a girl with almost complete unilateral ptosis. I noticed nothing peculiar about this, except that she had 20 degrees of right hypotropia. I performed a Blaskovics operation, and she returned satisfied with the result. However, she told me that her parents had observed that when she was eating the lid operated on opened more widely than did the lid of the other eye. I found that she had a Marcus Gunn phenomenon. Here was a patient whose ptosis was so marked that I did not see the Marcus Gunn phenomenon, but after the operation it was obvious that on biting down while eating she raised the lid about twice as high as the other lid; when she was not biting down, she looked quite well. The operation requires time, but if it is done according to Lindner's illustrations, which the operator should have before him the first time he attempts the operation, it is a satisfactory procedure, even in cases of practically complete ptosis with paralysis of the superior rectus muscle in which the Motais operation is not indicated.

DR. CONRAD BERENS, New York: During the past two years, at the New York Eye and Ear Infirmary a procedure somewhat similar to Dr. Jameson's (suggested by George Young) has been employed in which the entire superior rectus muscle is used. Young suggested drawing the

muscle down, as advised by Dr. Jameson. He then inserted three double-armed sutures in the muscle, passing these through the upper eyelid after excising a piece of the upper border of the tarsus.

DR. P. CHALMERS JAMESON, Brooklyn: I believe that there is no single operation that is suitable for every case of ptosis. I like to divide the cases into those in which the "heavy burden operations" should be done and those in which the degree of ptosis is slight. I have mentioned some of the operations which can be used to correct lesser degrees of ptosis. I believe that my operation, so far as the superior rectus muscle is concerned, can be used to give motion, but often one of what I term the "heavy burden operations," such as the Eversbusch procedure, is needed to supplement the operation in order to obtain the best results.

There is one thing I should like to emphasize, and that is gradation. Little has been said about gradation in operations for ptosis, and I think that it is essential. An ocular muscle is strengthened by shortening it, but often no attempt is made to strengthen the superior rectus muscle sufficiently to bear the additional burden of a heavy lid in the same way. I think that this can be done to great advantage. It is not fair to expect an attenuated passive muscle like the superior rectus muscle to assume a load four or five times greater than it is able to carry, and that accounts for failure in so many of these operations on the superior rectus muscle. A combination of procedures should often be used. The operation on the superior rectus muscle will give motion, but it will give no independent motion to the lid.

TRANSLATION OF TENON'S CAPSULE IN OPERATIONS ON THE OCULAR MUSCLES, WITH ESPECIAL REFERENCE TO POSTOPERATIVE DEVIATIONS WITH ADHESIONS BETWEEN THE MUSCLES AND THE EYEBALL. DR. CONRAD BERENS, New York.

A piece of Tenon's capsule is reversed and sewed to the episcleral tissue beneath the muscle. The results of the operation in 3 patients seem to have been satisfactory.

DISCUSSION

DR. JOSEPH M. KEELER, St. Louis: Dr. Berens speaks of reversing a piece of Tenon's capsule. I wonder whether he has ever tried to free the capsule at one side and then slide it under the muscle, in the hope of preventing the reformation of adhesions.

DR. P. CHALMERS JAMESON, Brooklyn: I believe that Dr. Beren's procedure is quite feasible and should be tried. Of course, one of the controversies which is frequently advanced concerning shortening and advancement is that action is obtained only from the most posterior adhesions. If this operative procedure will substitute a normal undergrounding between the muscle and the eyeball, it would enable one to secure the full effect and correction from the point of insertion of the muscle. As a matter of fact, I think that the posterior adhesion theory has been overexaggerated. If one adheres to this strictly, in virtually every operation that is performed on an ocular muscle the muscle is advanced or receded to the equator. A disadvantage of this procedure might be that the insertion of a sector of capsule under the muscle might provide no better gliding base than is usually left after a recession

or advancement, as the capsule, muscle and sclera in the freshened state would firmly agglutinate. The dissection and placement of the capsule would then be both harmful and unnecessary.

One of the objections to the Worth operation, as stated by the advocates of shortening, is that one does not obtain any more effect from this procedure by reason of the muscle acting from its posterior adhesion. Of course, transplantation of Tenon's capsule is not likely to be as readily applicable in cases in which there are adhesions anterior to the insertion of the muscle, but if it should be, it would overcome the foregoing objection.

DR. CONRAD BERENS, New York: Dr. Keller's question as to whether one could not slide Tenon's capsule beneath the muscle also suggested itself to me. I employed advancement and lateral displacement of Tenon's capsule recently on a child with exotropia who had previously been operated on and who had a stitch abscess involving the medial rectus muscle. In cutting into the tissues I found the muscle to be decidedly atrophic, and there was extensive fibrosis. The part of the muscle that was fibrosed and adherent to the sclera 15 mm. from the cornea was excised. Tenon's capsule was found to be greatly thickened beneath the muscle, where it assumed an oblique angle. Since the capsule was so thick and was not adherent to the muscle in this position, the piece of the capsule was brought forward instead of being excised. This patient is not presented here because the operation was performed only a week ago. At present the eye continues to diverge.

Dr. Jameson's question in regard to where the muscle pull is exerted is an important point. I have always believed that resection produced almost as good results as advancement because of the adhesions to the old insertion. However, this would be extremely difficult to prove. I believe that it may vary according to the degree of fibrosis of the muscle and the extent of the adhesions. Adhesions were found between the muscle and the eyeball as far back as 20 mm. in 2 cases and 15 mm. in 1 case. Other methods of attempting to correct the deviations had been employed, with unsuccessful results.

In reply to my inquiry as to whether he had employed this operation, Dr. Peter asked if I would not produce more adhesions with Tenon's capsule, and whether I would have difficulty with these adhesions, thereby limiting the effect of the operation. It is possible that some undesired effect may be produced. However, in 2 patients this defect, as well as the tendency for the reformation of adhesions between the muscle and the sclera, was apparently overcome.

In 1 patient only 4 diopters of hypertropia was found before operation, and the postoperative studies revealed 14 diopters. This vertical deviation might possibly be the result of traction by scar tissue following the excision of Tenon's capsule.

THE MECHANISM OF THE FORMATION OF THE AQUEOUS. DR. JONAS S. FRIEDENWALD and DR. ROBERT D. STIEHLER (by invitation), Baltimore.

Previous studies have shown that the ciliary body exhibits an irreciprocal permeability to water and methylene blue. The present study represents an attempt to discover the physical basis for this mechanism.

It is found that there is a semipermeable, electrically charged membrane between the stroma and the epithelium of the ciliary body. This membrane is permeable to neutral and acid dyes and impermeable to basic dyes. The ciliary stroma is capable of reducing methylene blue and thus converting it to neutral substance, while the ciliary epithelium oxidizes reduced methylene blue. The energy for the irreciprocal premeability of the ciliary body is derived from the difference in oxidation reduction potential between the ciliary stroma and the epithelium. It is postulated that water is transferred across the membrane by electro-endosmosis.

DISCUSSION

DR. F. H. VERHOEFF, Boston: I should like to ask Dr. Friedenwald and Dr. Stiehler if similar experiments have been made on the conjunctiva.

DR. JONAS S. FRIEDENWALD, Baltimore: We have no experiments on the permeability of the conjunctiva to report. There is, however, already some evidence in the literature that the cornea possesses irreciprocal permeability. This has been demonstrated by F. P. Fischer, but the mechanism in regard to the cornea has not been interpreted.

OCULAR CHANGES EXPERIMENTALLY PRODUCED IN THYROPARATHYROIDECTOMIZED DOGS, WITH SPECIAL REFERENCE TO INTRA-OCULAR TENSION AND BLOOD PRESSURE: A PRELIMINARY REPORT. DR. GRADY E. CLAY and DR. J. MASON BAIRD (by invitation), Atlanta, Ga.

In all animal experimentation on the thyroid and parathyroid glands, no mention has been made of the state of the intra-ocular tension and blood pressure.

Ten years ago patients were seen who had a low tension (below 13 mm. Schiötz) without any other ocular pathologic process. In many of these patients opacities of the vitreous, and later posterior subcortical lenticular changes, developed. In 2 patients detachment of the retinas developed. A study of these patients (to be reported later) showed them to have a low basal metabolic rate and low blood pressure, which was not in direct proportion to the intra-ocular tension, and no patient presented symptoms of myxedema. The authors, therefore, feel that there is an essential hypotensive ocular disease which is of as much importance as the much studied hypertensive disease, glaucoma. These clinical findings led to a study of thyroparathyroidectomized rabbits and dogs, which was begun in 1929.

At the time 10 rabbits were studied following parathyroidectomy; 5 rabbits survived long enough for a daily study of the intra-ocular tension, and all showed a drop of 10 mm. or more. Since 1933 60 dogs have been used, and on 47 of these the basal metabolic rate, the blood pressure, the calcium content of the blood and aqueous, the intra-ocular tension and changes in the vitreous and lens have been minutely studied. The animals showed a marked decrease in the calcium content of the blood after operation, with a tendency to an increase as they came under dietary control. With the administration of parathyroid extract, the rise was accentuated but did not reach a normal level. Study of the aqueous had given indeterminate results with certain significant figures.

It will be shown that a sudden and constant drop in the intra-ocular tension occurred in all dogs. Coincident with this there was a lowering of the blood pressure, but it was not as great in proportion as that of the intra-ocular tension.

Dogs kept on a sustaining calcium diet, and especially those given parathyroid extract, had a definite increase in blood pressure and basal metabolic rate. During tetany, both the blood pressure and the intra-ocular tension increased.

Opacities of the vitreous developed in the dogs having a hypotension of four weeks' duration, and in such dogs lenticular opacities are much more likely to develop. These opacities usually begin to appear after from two to three months, the suture lines showing the earliest involvement.

Thyroparathyroidectomized dogs which were fed thyroid extract showed no increase in the intra-ocular tension, and animals with most of the thyroid and one pair of parathyroid glands removed did not show such a marked decrease in intra-ocular tension.

Intra-ocular hypotension is produced in dogs by thyroparathyroidectomy. The parathyroid glands seem to be the chief causative factor for this phenomenon. It is thought that the lowering of aqueous volume or aqueous stasis is responsible for the opacities of the vitreous and the development of this type of cataract. This may explain cases of idiopathic detachment of the retina.

DISCUSSION

DR. JONAS S. FRIEDENWALD, Baltimore: I should like to ask what pupillary reactions occurred in these animals, and whether the authors are sure that the cervical sympathetic nerves were not injured.

DR. GRADY E. CLAY, Atlanta, Ga.: In answer to Dr. Friedenwald's question, no careful study of the papillary reflexes was made until within the past few weeks, and observations on 5 dogs showed that there was no disturbance of their pupillary reflexes. We therefore felt that there was no disturbance of the cervical sympathetic system to explain the lowering of the intra-ocular tension.

BIRTH INJURIES OF THE CORNEA AND ALLIED CONDITIONS. DR. RALPH I. LLOYD, Brooklyn.

The typical injury of the cornea at birth is the result of compression of the globe against the roof of the orbit by the end of the forceps blade. After all of the acute changes have cleared, the signs of such an injury in a typical case are a high degree of amblyopia, a corneal scar, tears in Descemet's membrane and, after some time, keratoglobus. The damage to Descemet's membrane is most common and permanent and consists of either a series of concentric or parallel tears or a complete stripping of this membrane from the substantia propria. In the latter case there are also lacerations which separate the detached membrane into broad strips that run from attachments near the periphery across the anterior chamber free in the aqueous like the string of a bow.

This highly elastic tissue also rolls up from each lateral margin, converting each strip into a rod, which is almost transparent. This is one type of so-called "glass membrane in the anterior chamber." In

cases of long continued or active keratitis, folds appear in Descemet's membrane, and fibrin collects along these on the posterior surface of the cornea, forming another type of glass membrane. This type occurs most frequently in cases of interstitial keratitis.

During the course of sharp tuberculous iridocyclitis, another type of glass membrane is formed. Its appearance suggests that it is a defense mechanism for the protection of the cornea from the damaging effects of the deposits constantly thrown against this surface during the course of such inflammations and that they are really vascular in nature. The name of "posterior pannus" has been suggested for this type of glass membrane. The peculiar form of some of the glass membranes which develop in cases of interstitial keratitis is much like some of the remnants of the persistent pupillary membrane attached to the posterior surface of the cornea.

DISCUSSION

DR. THEODORE L. TERRY, Boston: In 1926, before the Ophthalmological Society of the United Kingdom, Ballantyne reported a case of detachment of Descemet's membrane resulting from a birth injury inflicted by forceps. He was able to examine the eye again when the child was 5 years of age. By oblique illumination he found a somewhat crescentic figure in the upper portion of the cornea. The upper line was somewhat serrated, whereas the lower line formed a gentle curve. With the slit lamp he discovered an apron-like detachment of Descemet's membrane, the upper wavy line being the base and the lower curved line being the free edge. In the beam of the slit lamp at the lower edge of the detachment there was a bright, expanded bead. The shape of this detachment no doubt prevented extensive rolling up of Descemet's membrane into a rod, as Dr. Lloyd has observed.

At the laboratory of the Massachusetts Eye and Ear Infirmary there are sections of just such an eye. The original central core of Descemet's membrane hangs down into the anterior chamber, on which there are several newly deposited layers of this membrane. Since the free edge of the membrane has rolled somewhat, the newly formed Descemet's membrane covers over this roll, producing a nodular extremity. Such an extremity would give the appearance of a bright bead in the beam of the slit lamp. Presumably, the layers of the newly formed Descemet's membrane are due to repeated loss and regrowth of endothelium. This patient noticed slight irritation and redness of the eye at the age of 21. She consulted Dr. Verhoeff, who observed recurring attacks of bullous keratitis. There was no history of birth injury or trauma to the eye. Finally, glaucoma developed, and since the vision had always been poor, the eye was removed.

CYCLOPLEGIA WITH BENZEDRINE AND HOMATROPINE: PRELIMINARY REPORT. DR. S. JUDD BEACH and DR. W. R. MCADAMS (by invitation), Portland, Me.

The work of Myerson, showing the synergistic action of atropine and benzedrine so that minimal doses control the ciliary action, suggested the possibility of combining benzedrine with homatropine for the same purpose. In a series of adults it was found that cycloplegia followed in one hour after a single instillation of benzedrine and homatropine.

Ability to read returned in many cases as early as six hours after the instillation. By using the new combination in one eye and homatropine alone in the other eye, it has been possible to compare the efficiency. The added safety of the transitory mydriasis is obvious.

DISCUSSION

DR. ALAN C. WOODS, Baltimore: I should like to ask Dr. Beach and Dr. McAdams if any further observations have been made on the same eye on the amount of cycloplegia obtained with a combination of benzedrine and homatropine and later on the amount of cycloplegia obtained by the use of atropine.

DR. C. A. CLAPP, Baltimore: I should like to inquire whether any patients have shown an idiosyncrasy to this drug.

DR. PARKER HEATH, Detroit: Were any changes in the intra-ocular pressure noted?

DR. S. JUDD BEACH, Portland, Me.: Our observations are not yet complete enough to give satisfactory answers to most of these questions. As Dr. Woods suggests, we have used the two drugs separately at different intervals, but we have no data on this point worth considering. We have not yet encountered any idiosyncrasies.

INTRA-OCULAR INVASION BY THE LARVA OF THE ASCARIS: REPORT OF A CASE WITH UNUSUAL COMPLICATIONS. DR. F. PHINIZY CALHOUN, Atlanta.

This paper was published in full in the December 1937 issue of the ARCHIVES, page 963.

TREATMENT OF OCULAR TUBERCULOSIS. DR. ALAN C. WOODS and DR. M. ELLIOTT RANDOLPH (by invitation), Baltimore.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 510.

DISCUSSION

DR. FRANCIS H. ADLER, Philadelphia: I should like to ask Dr. Woods and Dr. Randolph if they have any data that show a correlation between the clinical improvement in their patients and the loss of cutaneous sensitivity. I am in accord with the theory that allergy and immunity are two separate factors, and on that basis the patient who shows a marked cutaneous sensitivity should also show some correlation between his clinical improvement and the loss of cutaneous sensitivity after a course of treatment with tuberculin. Unfortunately, in my hands at least, I have been disappointed in that they do not seem to go hand in hand. The patient who has a marked cutaneous sensitivity is not always the one who does best with tuberculin therapy. Sometimes the patient who has a low cutaneous sensitivity is the one who shows most marked improvement, and, furthermore, when clinical improvement occurs after a course of tuberculin, the patient who has a high cutaneous sensitivity still maintains it.

I believe that every ophthalmologist should use some standardized technic in his cases. There is now a standardized purified derivative of

tuberculin which can be used for testing cutaneous sensitivity. Often the figures in the literature are confusing, largely because of different tuberculins which have been used, but every one can use this purified protein derivative and obtain results which should be uniform.

DR. F. H. VERHOEFF, Boston: As I understand it, Dr. Woods and Dr. Randolph have taken a series of cases of ocular tuberculosis of various kinds—hardly any two cases can be compared with each other, since they are so different—and have treated one group of patients with tuberculin by one method and another group by another method, but I do not understand that they have a control group to whom tuberculin was not given. For the sake of argument, if I should say that their results simply show that one of the methods of treatment with tuberculin made the patients a little worse than if tuberculin had not been used and that the other method made them even more worse, how could they deny this? My impression is that a control series of patients to whom tuberculin is not administered gives a good set of results. Moreover, if one has a large control series and divides the patients indiscriminately into two groups, I am sure that the results in each group will differ considerably. I am simply pointing out how difficult it is to draw conclusions from statistics, especially when one is dealing with cases that are individually so different.

DR. ARNOLD KNAPP, New York: I belong to the group that believes that tuberculin therapy helps ocular conditions. If I understood correctly, Dr. Woods and Dr. Randolph have undertaken treatment with tuberculin along two lines: first, by using increasing doses and, second, by using the small doses over a long period. I should like to ask how they differentiate the cases before administering treatment—what type of ocular tuberculosis requires which of these two forms of treatment? I have carried out both types of treatments, and both have given good results. The second, or the one in which an "optimum" dose is used, has given the best results, which agrees with Dr. Woods and Dr. Randolph's experience. In using the latter method, I have been much pleased with the use of A O tuberculin.

I was particularly interested in hearing the report on the beta ray radium, and I hope that Dr. Woods will go into particulars a little more fully in his closing remarks.

DR. E. V. L. BROWN, Chicago: I sympathize with Dr. Verhoeff's side of this question. I should like to see more series of patients who are not treated, because the disease tends to be self-limited.

I want to refer to a series of 4 cases that I reported in St. Louis last year and that were later reported in the *American Journal of Ophthalmology*. In these there was a parallelism between the tuberculosis of the anterior segment and the definite findings in the chest. The condition was of marked severity in each case. From 10 to 12 nodes were present in each iris, and these left Michel flecks in the irides after the nodes were absorbed; the keratitis consisted of a glomerular-form arrangement of vessels, with deep nodes in the cornea. Treatment with tuberculin was not effective. All the patients were cured by thorough rest. I believe that there is a growing number of cases in which there is a parallelism between the nature and the severity of the findings in the chest and those in the eye. I have had another case this year in which a massive

nodular tuberculosis of the iris was present. There were no lesions in the chest for many months, but in the past two months there has been a marked development of pulmonary lesions.

Dr. Alan C. Woods, Baltimore: Dr. Adler has put his finger on the crux of the question so far as sensitivity in ocular tuberculosis is concerned. There is no exact information on the relation of ocular sensitivity to cutaneous sensitivity. In ocular tuberculosis one is chiefly concerned with the question of the sensitivity of the eye. We have no means of testing this, and we therefore test the cutaneous sensitivity on the theory that cutaneous and ocular sensitivity may run parallel. For the past year this question has been under active investigation in the Wilmer Institute. Our experiments, the first of which will soon be reported, indicate that under normal conditions cutaneous and ocular sensitivity may run parallel. Other conditions, notably ocular tuberculosis, may radically alter this normal parallelism. A study of this relationship may affect our belief as to the time tuberculin therapy should be pushed or omitted. It may be shown that it should not be pushed during the periods of ocular activity, when the patient is secreting tuberculin in his own eye. The relationship of ocular sensitivity to cutaneous sensitivity in the various stages of ocular tuberculosis must be determined.

I should like to make one comment about purified protein derivative. This substance is valuable only for diagnostic purposes, and in the Phipps Clinic, in Philadelphia, the fact has been established that it is nonantigenic and that one cannot obtain desensitization with it. To obtain desensitization, some type of tuberculin itself must be used and not purified protein derivative. The essential point in the choice of tuberculin is that one use a standardized tuberculin of known activity.

So far as Dr. Verhoeff's criticism goes, we have no control series of patients with ocular tuberculosis and probably never shall have. Certain patients have not been treated, owing to active pulmonary complications or because of other contraindications. Since we are convinced of the value of tuberculin in ocular tuberculosis, we do not feel justified in withholding it from a series of patients whom we feel it would benefit. Hartig, in Germany, had a control series, and of his 14 patients from whom he withheld tuberculin he observed spontaneous improvement in only 1.

In regard to Dr. Knapp's question of the choice and dosage of tuberculin, as has previously been stated, one must have a tuberculin of known and standardized strength. There is no such thing as an absolute dose of tuberculin; the dosage must be regulated according to the reaction of the patient. We have abandoned, so far as possible, the perifocal plan of treatment and now usually treat all our patients on the basis of the desensitization theory. Here the essential point is to keep the dose just below the patient's point of reactivity. This requires constant observation and may be an exceedingly delicate and difficult thing to do.

Dr. Brown's question as to the relation of the general tuberculous status of the patient to the ocular activity is a pertinent one. Werdenberg insists that all patients with ocular tuberculosis exhibit definite roentgenographic evidence of pulmonary changes. In a great many of our cases we had roentgenograms made. That phase of the subject was not

discussed in this paper. I agree with Werdenberg that ocular tuberculosis is usually secondary to tuberculosis of the hilus glands, but I cannot agree that one can always demonstrate these changes. Certainly the American radiologists must read as negative pictures regarded by Werdenberg as showing slight fibrotic changes. We took the question up with Dr. Pierson, the head of the roentgenologic department at Johns Hopkins Hospital and University. We examined many old plates that were interpreted as negative, and undoubtedly they frequently showed low grade hilus shadows. Dr. Pierson takes the view that such changes are normally seen in adult patients and that the roentgenographic picture should therefore be considered negative. In our present series of cases we are attempting to correlate the exact changes in the hilus gland with the ocular tuberculosis.

FORMATION OF PRERETINAL CONNECTIVE TISSUE IN ACUTE CHOROIDITIS: REPORT OF THREE CASES. DR. ARNOLD KNAPP, New York.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 558.

PATHOGENESIS OF DISCIFORM DEGENERATION OF THE MACULA. DR. F. H. VERHOEFF, Boston, and DR. HERMAN P. GROSSMAN (by invitation), Providence, R. I.

This paper was published in full in the October 1937 issue of the ARCHIVES, page 561.

DISCUSSION

DR. ALBERT N. LEMOINE, Kansas City, Mo.: The observations of Dr. Verhoeff and Dr. Grossman on the pathogenesis of disciform degeneration of the macula are exceedingly important to the ophthalmologist. These findings are not necessarily limited to lesions in the macula, but point to the possible pathogenesis of similar lesions elsewhere in the retina.

Now that the pathogenesis of these lesions has been demonstrated, with my observations as to the etiology of some of the intra-ocular capillary hemorrhages, it is highly possible that a number of these are due to vitamin C deficiency or mild scurvy. When there is increased capillary permeability sufficient to cause hemorrhages in the retinal vessels or choriocapillaris, one must think of arteriosclerosis or cardiovascular renal disease. However, one must not lose sight of the fact that some internists attribute some of those conditions to vitamin C deficiency. This deficiency might be due either to lack of assimilation or to intake of the vitamin C.

There is enough evidence to point to the possibility of some capillary intra-ocular hemorrhages being due to vitamin C deficiency to justify making the tourniquet test in all cases of idiopathic hemorrhages, and if such tests are made and the reactions are found to be positive, to administer cevitamic acid.

DR. ARNOLD KNAPP, New York: I am glad that Dr. Verhoeff and Dr. Grossman have had the opportunity of examining some patients with disciform degeneration of the macula and of elucidating the pathologic picture. I have been interested in this condition since 1919, when

I presented before this society a case in which I made a diagnosis of sarcoma. I should like particularly to speak of the clinical differentiation between a tumor and an extravasation of blood in the macular region. Disciform degeneration occurs, in my experience, in older persons; the elevation is never more than 3 or 4 diopters; the outline is fairly round or oval, and the mass is amorphous either in the affected eye or in the other eye and usually gray or grayish white. There is definite evidence of arteriosclerosis. The occurrence of discoid macular degeneration in association with circinate retinitis was mentioned. While macular changes are frequent in cases of circinate retinitis, they are not like those described in this paper.

Dr. Theodore L. Terry, Boston: I should like to report a case in which a small macular lesion of the right eye in a woman 29 years of age had the appearance of a malignant melanoma and produced a central scotoma. Dr. Beetham had the patient examined by several ophthalmologists before he removed the eye. The lesion proved to be typical disciform degeneration with associated hemorrhage and serum. In the choroid itself, subjacent to the disciform lesion, there was a nodule of chronic inflammatory cells. Another somewhat similar inflammatory lesion was situated near the nerve head.

Unfortunately, this case was brought to the attention of Dr. Verhoeff and Dr. Grossman too late for them to be able to include it in their paper. Dr. Verhoeff has studied these slides, and I should like to hear his interpretation of the findings.

Dr. Herman P. Grossman, Providence, R. I.: Dr. Knapp has presented a few points in the diagnosis of senile disciform degeneration of the macula, particularly as to the differentiation of this condition from sarcoma of the choroid. This differentiation is based on: (1) the shape and contour of the lesion, (2) the elevation of the mass and (3) the associated presence of retinal arteriosclerosis in disciform degeneration.

In many cases reported in the literature the contour of the lesion was not at all disciform. We are convinced that the configuration of the lesion is not helpful in the diagnosis. The height of the tumor is of little significance. The variation of elevation in the reported cases ranged from 0.5 to 6 diopters. Sarcoma in its later stages no doubt reaches a much greater height, but at that late stage this sign is no longer needed for confirmation of the diagnosis. In a summary of the literature retinal arteriosclerosis was mentioned in only 25 of the 129 cases. Retinal angiosclerotic vascular changes were found histologically in only a few of the eyes which were examined microscopically. These figures are, of course, of no significance whatever in a group of patients whose average age is 68. One can therefore place little reliance on this sign as an aid in differentiating disciform degeneration from sarcoma.

We believe that the important points in the differentiation of the conditions are these: Typical subretinal hemorrhages adjacent to the lesion are seldom if ever seen in cases of sarcoma. The location of the tumor is important, since sarcoma has no particular predilection for the macular region and seldom occurs at that point, whereas disciform degeneration always includes the macula.

Finally, in cases of sarcoma separation of the retina is very common and often appears to be entirely out of proportion to the size of the

tumor. On the other hand, in cases of disciform degeneration separation of the retina, when present, is slight and is often not sufficient to be recognizable by ordinary ophthalmoscopic investigation.

DR. F. H. VERHOEFF, Boston: Dr. Lemoine's observations are interesting. There must, of course, be a great variety of causes of petechial hemorrhages, and vitamin deficiency may be one of them, but we have no evidence of that in our cases. We know that subconjunctival hemorrhages occur at times without the cause ever being detected. No doubt, in the majority of the cases of senile disciform degeneration the hemorrhage results from an arteriosclerotic process. In some of these cases there are hemorrhages away from the lesion, but I think most of these hemorrhages are really blood that has extended under the retina from the main mass. In patients with arteriosclerosis there may be retinal hemorrhages independent and separate from the main mass.

Dr. Knapp stated that he believed that there was no connection between disciform degeneration of the macular region and circinate retinitis. A number of cases of circinate retinitis associated with this condition have been reported in the literature. I recently saw 2 cases in the clinic. In 1 there were circinate lesions in the retina over the mound. The relation of the two conditions is described in our paper.

In Dr. Beetham's case, referred to by Dr. Terry, it was evident the small lesion in the macula was of the same nature as those in the senile type of disciform degeneration. I found blood still present in the mound and also blood around it. The striking thing in this case was the marked lymphocytic infiltration of the choroid behind the mound. I saw Dr. Knapp's specimen (he sent a section to me at the time), and I have seen sections of the lesions in 5 other cases, and in none of them was there such marked lymphocytic infiltration.

In Dr. Beetham's case there was also lymphocytic infiltration at the margin of the disk entirely independent of the other, so it would seem that there must have been a low grade metastatic infection. Here is an instance of a disciform lesion in a young person similar to those occurring in elderly persons. The patient was only 29, and the lesion never could have subsided without marked impairment of vision resulting. In this way it was also like the senile type of lesion. In this case, however, the cause of the hemorrhage was different, no doubt. The patient did not have arteriosclerosis. There are probably a great many conditions which lead to hemorrhage from the choriocapillaris. All I wish to point out is that a typical lesion is due to organization of a hemorrhagic extravasation which is primarily beneath the pigment epithelium. In some cases the hemorrhage may be due to urticaria. I can even bring my favorite factor, allergy, into the question. It is useless, however, to discuss all these points unless one has definite evidence of them.

THE GENESIS OF THE CYCLITIC MEMBRANE. DR. HARVEY D. LAMB, St. Louis.

The cyclitic membrane formed at the inner free surface of the ciliary body is an example of productive fibrosis. It originates in association with chronic cyclitis, in which the most constant symptom is an exudation of macrophages into the adjacent vitreous. At the latter site, the macro-

phages become fibroblasts and form the cyclitic membrane. Some examples of this incipient formation are described and illustrated with photomicrographs.

NEUROGENIC ORIGIN OF CHOROIDAL SARCOMA. DR. GEORGIANA DVORAK-THEOBALD, Oak Park, Ill.

This paper was published in full in the December 1937 issue of the ARCHIVES, page 971.

INFLAMMATORY PSEUDOTUMOR OF THE ORBIT: REPORT OF A CASE. DR. PHILIP M. LEWIS, Memphis.

A case of pseudotumor of the orbit with satisfactory surgical removal is reported.

DISCUSSION

DR. C. A. CLAPP, Baltimore: In discussing this paper I wish to report a case that is almost identical in history and appearance. It occurred in a man of about 40, who first noticed beginning proptosis in July 1936. I first saw him in September 1936, at which time he had 4 mm. of proptosis with limited motion but normal visual fields. The proptosis increased, however, and even in the presence of a negative Wassermann reaction he was treated with both mercuric and potassium iodide, without result. He was also given irradiation therapy, which was likewise without result. The proptosis increased until it measured 8 mm. and hemorrhages began to appear in the fundus. As consultants agreed with my opinion that an orbital growth was present, and in view of the severe pain, dissection was made above, and the tumor, or pseudotumor, was found to extend clear to the optic foramen. A frozen section was made, and "suspected malignancy" was reported. The tumor, or growth, including the apex of the muscle cone, was removed. The photomicrograph of the tissue is similar to that in Dr. Lewis' case. There has been considerable discussion as to the diagnosis. Dr. Rich, of the pathologic department, is of the opinion that it is an inflammatory growth, and Dr. Friedenwald believes that it is syphilitic. In view, however, of the repeated negative Wassermann reactions, and since the patient showed no improvement on antisyphilitic treatment, I doubt very much, at least from the clinical aspect, whether this growth was of syphilitic origin.

DR. ALGERNON B. REESE, New York: I should like to cite a case of pseudotumor of the orbit with some unusual features. The woman had a proptosis of the right eye, and through the upper and lower lids, at the inner canthus, a mass was palpable. A biopsy specimen, secured by aspiration, showed the usual chronic inflammatory granuloma. In the course of six months the eye receded, and slight enophthalmos ensued. After six months the left eye protruded, and through the lower lid, at the external canthus, a mass was palpable. The exophthalmometer readings showed that the left eye was 6 mm. more prominent than the right. This difference between the two eyes was not due entirely to the exophthalmos on the left side but also to the enophthalmos on the right side. After one month the difference in the two eyes was only 2 mm.; in another month it was only 1 mm., and three months later there was

an enophthalmos of the left eye. The eyes remained the same for two years more, and then the left eye again protruded 5 mm. At the same time there appeared a large, smooth, somewhat firm mass over the forehead, and a biopsy specimen secured by aspiration showed that it was a chronic inflammatory granuloma. After two months the exophthalmos began to recede, until it was only 4 mm. In two months more it was only 1 mm. Six months later there was an enophthalmos of 2 mm.; that is, the exophthalmometer reading on the left eye was 15 and on the right eye, 13. The only possible indication of the etiology was that the roentgenographic examinations showed an elevation of the outer table of the frontal bone, and there was a denseness in both antrums, thus suggesting the possibility that an inflammatory granuloma had extended from the sinuses into the orbit and over the frontal bone.

CYST OF THE POSTERIOR CHAMBER. DR. ERNEST F. KRUG, New York.

A case is reported in which a cyst of the posterior chamber developed nine years after injury to the globe and was treated by electrocoagulation. It reappeared after eighteen months, and the electrocoagulation was repeated. Two months later the cyst again reappeared with secondary glaucoma, necessitating removal of the eyeball.

DISCUSSION

DR. F. H. VERHOEFF, Boston: Apparently it is technically correct to speak of this growth as a cyst of the posterior chamber, although the cyst did not originally occupy the posterior chamber. It is unfortunate that this title had to be used, because it gives a wrong impression, namely, that the case differs from that of a cyst in the anterior chamber following a perforating wound. I can recall 3 cases in which I have operated for epithelial cyst of the anterior chamber. The results were successful in all 3.

The point I wish to make is this: In such cases it is well worth while to attempt to remove the cysts, even if one does not succeed in excising all the epithelium. I believe that the eye can harbor a certain amount of it: possibly as a result of the operation the epithelium is incarcerated in scar tissue so that it does not proliferate.

DR. THEODORE L. TERRY, Boston: That Dr. Krug has used the term "epithelial cyst of the posterior chamber" is of some importance, because it shows that an implantation epithelial cyst can extend into the posterior chamber.

In examining the sections in the laboratory of the Massachusetts Eye and Ear Infirmary, 2 instances of epithelium in the posterior chamber were found, a coloboma of the iris being present in each. No case of epithelial cyst in the posterior chamber was discovered, however.

In Dr. Krug's case it is interesting to note that the epithelium lining the cyst varies greatly in the number of its layers. Although this is not necessarily important, the fact that the epithelium is reduced to one or two layers in that region of the cyst exposed to the aqueous and that the central area of this free portion shows necrosis of the epithelium may indicate that the aqueous does not bring it sufficient nutrition or that there is some growth-retarding factor in the aqueous. This behavior

of the aqueous, I believe, has been suggested by those who are particularly experienced in corneal grafting. This is further borne out by the fact that in other localities it is possible to find blood vessels in contact with the cyst.

In view of Dr. Verhoeff's observations, attempted removal would apparently be a worthwhile procedure; however, the size and nature of this cyst would have made complete removal difficult or, more probably, impossible.

DR. ARNOLD KNAPP, New York: May I speak on the question of treating a cyst of the iris by electrocoagulation? The patient I wish briefly to refer to was a woman operated on for a cataract a year before I saw her, and when she came to me she had a broad adhesion of the iris and of the capsule to the section, a well developed cyst in the iris and increased tension. The cyst was treated by electrocoagulation in the usual manner, and the result was very favorable for eight months. After that time the tension returned; it was difficult to tell whether the return of the tension was due to the adhesion of the iris and capsule or to a return of the cyst. In any case, cyclodialysis was done. This made matters worse, and the eye gradually became phthisic. I do not feel that electrocagulation is of great help in these cases, nor do I believe that a cyst like this could be removed by an operative procedure.

DR. P. CHALMERS JAMESON, Brooklyn: Dr. Krug's interesting case recalls one that I saw a year ago. About twenty years previously a young boy had both eyes injured by an explosion. I had to enucleate one eye, which was severely wounded, and extract the lens of the other, which had a perforating wound of the iris. About twenty years later, i. e., last year, a large cyst developed, extending from the lower limbus well into the small pupil which I had retained for him. All through these years he had maintained vision of 20/30. I was undecided as to what treatment to pursue.

At a meeting of the New York Ophthalmological Society a case was reported in which a solution of iodine had been injected. The technic was interesting. A few drops of tincture of iodine was removed in a pipet; the fluid from the cyst was withdrawn and allowed to mix with the tincture of iodine; the mixture thus obtained was injected into the cyst before the needle was withdrawn. In this case there was no reaction, and a perfect result was obtained.

My own surgical experience in removing these cysts has not been successful. I have not done many of these extirpations, but the cysts have nearly always recurred. The result so far as the destruction of the cyst was concerned was satisfactory, but later a condition developed which resembled multiple tubercle nodules. I did everything I could, and yet the pupil is occluded, and an active inflammatory condition has continued over the past year.

With regard to the suggestion relative to electrocoagulation, one notes that in the 2 cases just reported glaucoma developed later. While electrocoagulation destroys the cyst itself, is it not possible that the amount of heat required and generated to destroy the cyst may impair the tissues at the filtration angle so that a glaucoma would later develop? That is the consideration which would possibly prevent me from using

electrocoagulation in this region, and as surgical treatment is not always successful, one may have to rely on the injection of chemical solutions to destroy the walls of the cyst.

Posterior Sclerotomy as a Form of Treatment in Subchoroidal Expulsive Hemorrhage. Dr. Derrick Vail, Cincinnati.

Two cases are reported in which it was felt certain that posterior sclerotomy saved the eye and prevented complete deterioration of vision.

The first case was that of a woman aged 41 who had undergone an operation for the removal of cataract from the right eye. The next day, while the wound was being dressed, typical symptoms of subchoroidal hemorrhage occurred, with a gaping wound. A Graefe knife was plunged into the temporal side of the eyeball, about 14 mm. from the limbus, leading to the appearance of bright red blood and the subsidence of pain within a relatively short time. Later the coagulated blood, tangled iris and coagulated vitreous between the lips of the gaping wound were excised, and the cornea was sutured to the sclera. About six weeks later iridectomy was performed, and the vision eventually was 20/25, with correction.

The second case was that of a woman of 65 who had noninflammatory glaucoma of the right eye following iridectomy with a Graefe knife; choroidal symptoms of expulsive subchoroidal hemorrhage appeared. An incision in the temporal side of the eyeball resulted in the appearance of bright red blood, which flowed for about two minutes, at the end of which time the pain was less, but still severe. Convalescence from then on was uneventful. The tension at the time of the patient's discharge was entirely normal, and the vision, which had been 20/25, was 20/200. In both cases there was a white streak in the choroid on the temporal side radiating up as far as could be traced anteriorly.

DISCUSSION

Dr. Bernard Samuels, New York: Dr. Vail has requested me to base my discussion on an examination of three of his lantern slides.

The first slide represents a typical nonexpulsive subchoroidal hemorrhage. The difference between this type and the ordinary expulsive hemorrhage consists in the fact that in the nonexpulsive hemorrhage there is no rupture in the choroid, but in the expulsive hemorrhage, in which the eye is always lost, the choroid ruptures and almost the entire contents are extruded. The nonexpulsive hemorrhages are nearly always more marked on the temporal side. The retina is never detached, because the retina and choroid are pressed together against the vitreous. These subchoroidal hemorrhages take place far forward, and posteriorly they seldom reach to the margin of the optic nerve, leaving a considerable zone surrounding the optic nerve.

The second slide is that of a glaucomatous eye in the horizontal meridian. The eye was not removed until ten days after iridectomy. Nonexpulsive hemorrhages do not always occur just at the time of the operation but may take place a number of hours afterward. In this respect they differ from the ordinary serous choroidal detachments described by Dr. O'Brien before this society. A serous detachment occurs slowly, because it takes a much longer time for transudate to

accumulate than for a hemorrhage. In serous detachment the globe is soft, whereas in nonexpulsive subchoroidal hemorrhage the blood accumulates rapidly. In the latter cases the eyeball is hard.

The third globe is cut in the vertical meridian, taking in the site of a trephine operation. At the end of the operation the vitreous presented itself in the wound. The eye was not enucleated until some days later. The blood in the subchoroidal space is remarkable because it is so well preserved. It shows how difficult it is for the choroid to become detached in the neighborhood of the veins of the vortex. The hemorrhage is the result of a rupture in one or both of the long posterior ciliary arteries. Most of the ruptures of the blood vessels seem to occur on the temporal side, and it is on this side that there is the greatest accumulation of blood.

According to laboratory experience, nonexpulsive subchoroidal hemorrhage occurs more frequently after the trephine and the Lagrange operation than after simple iridectomy. The explanation is that after these operations the globes remain soft for a long time. Whereas the diseased blood vessels might withstand a reduction in tension for a short period, they would be more likely to rupture if the reduced tension continued over a prolonged period.

CORRECTIONS

In the article "Technic of Goniotomy" by Dr. Otto Barkan in the February issue (ARCH. OPHTH. **19**: 217, 1938), the sentence beginning with the word "Reoperation" in the tenth line of the first paragraph on page 223 should read: "Reoperation in these cases will presumably normalize tension without the use of miotics. In a few cases in which the trabeculum was missed or the canal was not opened there has not been any effect on the intra-ocular pressure and reoperation will be required."

In the article by Drs. Abraham Myerson and William Thau entitled "Human Autonomic Pharmacology: IX. Effect of Cholinergic and Adrenergic Drugs on the Eye" (ARCH. OPHTH. **18**: 78 [July] 1937), "mydriasis" should be substituted for "miosis and cycloplegia" in the last line.

Book Reviews

Die Funktionsprüfung des Auges unter besonderer Berücksichtigung der Störungen des Farbensinnes. By Prof. Dr. Hans Oloff and Dr. Hans Podestà. Price, 7.50 marks. Pp. 198. Berlin: Julius Springer, 1937.

This book includes a second, enlarged and completed edition of the "Diagnostik der Farbensinnstörungen" of Stargardt and Oloff, which appeared in 1912.

The examination for color vision is a problem not well understood by many oculists. Hence, an exhaustive description of this examination is given, occupying more than half the text, and other tests for vision are added. These consist of the examination of vision, of light sense and of ocular motility. The main reason for these tests is that they concern safety in traffic, and only those tests are described which have proved to be essential, easily applied and reliable.

The part of the text dealing with the examination of color vision follows a chapter on the examination of visual acuity and on the chief refractive anomalies. The importance of color vision in one's daily life and activity is dwelt on. The exact determination of the type of defect in color vision is of secondary importance, except for scientific study and in the selection of certain professions. The tests for color vision can be made only by properly trained physicians. The defect cannot be relieved by any treatment and remains a congenital and transmissible condition. The material on the history, the development of color sense, the frequency of defects and its inheritance is followed by an analysis of normal color vision, the grades of color perception, the laws of spectral color mixtures, the theories of color perception and sections dealing with the color vision of the dichromats, the abnormal trichromats, color asthenopia, total color blindness and the acquired disturbances of color vision.

Diagnosis of disturbances of color vision is made with spectral light; samples of pigment, which include Holmgren's wool; colored crayons; pseudo-isochromatic charts; colored signal lights, and lanterns. The field of vision, light sense, motor anomalies, stereoscopic vision and simulation are also described. The German requirements for the army and the navy, for aviation and for railroads and motor vehicles are given in a concluding chapter.

This is an excellent manual, and the chapters on color vision and its disturbances are particularly worthy of praise. The text is clearly written and the printing is good, but it seems a pity that all illustrations have been omitted.

ARNOLD KNAPP.

Zur Therapie der Embolie der Zentralarteria der Retina. By Dr. Ernst Johansson, Zeitschrift für Augenheilkunde, supp. 24. Price, 6.40 marks. Pp. 86. Berlin: S. Karger, 1937.

After a brief description of the clinical picture known as embolism of the central retinal artery, it is stated that the cause is generally to be found in lesions or disorders (1) of the heart and vessels

(2) of the endothelium and (3) of the blood. It has been found that the sinking reaction of the erythrocytes is increased. A prophylactic agent containing an injectable extract prepared from fresh liver (camponol) has been found of value in some cases. Intravenous injections of eupaverin have been used successfully to relieve the spasm; the action of the drug was found to be of three hours' duration, and it must be given during the first ten hours. A good therapeutic agent consists of eupaverin 0.03 Gm., atropine methylbromate 0.0003 Gm. and aminopyrine 0.15 Gm., which is given in tablets and suppositories. Many forms of treatment are described, with references to the literature, and brief histories are added. Of 103 cases in which healing or improvement occurred, massage was successful in 30; operation in 27 (paracentesis in 16); blood letting, the administration of digitalis, the injection of a pancreatic preparation (angioxyl) and of atropine in 4 each, and the administration of acetylcholine in 7, of amyl nitrite in 2 and of strychnine and nitrites in 1, while spontaneous recovery took place in 16. In the last three years the author has observed and treated 9 patients with arterial closure, and the details concerning them are given. In conclusion, the following treatment is recommended: the injection of eupaverin, vibratory massage, the administration of padutin, the injection of atropine the administration of acetylcholine or operation (paracentesis). Prophylactically, the injection of the extract prepared from fresh liver is suggested.

ARNOLD KNAPP.

Manual of the Diseases of the Eye. By Charles H. May, M.D. Fifteenth edition. Price, $4. Pp. 498, with 376 original illustrations, including 25 plates, with 78 colored figures. Baltimore: William Wood and Company, 1937.

The new edition of this well known manual has been thoroughly revised, with the assistance of Dr. Charles A. Perera.

The chapters on the ophthalmoscope and on the ocular manifestations of general diseases have been rewritten; changes and additions have been made in the description of operations on the lids and for detachment of the retina. Additions have been incorporated, such as those dealing with dinitrophenol cataract, inclusion bodies, inclusion blenorrhea, acetylcholine therapy, "floaters," gonioscopy, the use of (paranormobutylaminobenzoyldimethylaminoethanol) pontocaine hydrochloride, recumbent spectacles and polaroid glass. Some new illustrations have been added, and others have been replaced.

Though the volume has been thoroughly brought up to date and many improvements have been added, it has not been increased in size. It continues to be one of the best textbooks for the undergraduate student and for the general practitioner.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Netherlands.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. I.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Plymouth. Time: July 20-22, 1938.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: July 4-6, 1938.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Place: Memorial Ophthalmic Laboratory, Giza. Time: March 25, 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 7-9, 1938.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31 E. Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: San Francisco. Time: June 9-11, 1938.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Gordon M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. C. Gardner, 11 N. Main St., Fond du Lac.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Marshfield. Time: May 1938.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Victoria, B. C. Time: June 21-24, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown, Pa.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Johnstown, Pa. Time: May 19, 1938.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. R. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. Rosenberger, 221–5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514–6th St., Valley City.
Place: Bismarck. Time: May 1938.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165–8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. D. Carroll, 102—3d St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606-20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: A. Lloyd Morgan, Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queen's Pk. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.